A DISSERTATION ON

ANALYSIS OF ETIOLOGICAL CAUSE FOR PATIENTS PRESENT WITH FEVER AND JAUNDICE AND ANALYSE CLINICAL PROFILE OF THOSE WHO HAVE LEPTOSPIROSIS

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BONAFIDE CERTIFICATE

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INTRODUCTION

Short duration of fever with jaundice is one of the common clinical scenarios that we come across in medical wards. Most common etiological agent responsible for such clinical scenarios are **Leptospirosis, Malaria and viral hepatitis.**

Other uncommon causes like Cholangitis, Sepsis, Dengue fever, Enteric fever, viruses like Human Immunodeficiency Virus (HIV), Epstein-Barr Virus (EBV), Cytomegalo Virus (CMV), and Varicella - Zoster Virus (VZV). Other non-infective causes like alcoholic hepatitis, drug-induced hepatitis and haemolytic uremic syndrome also presents with fever and jaundice.

The pattern of etiological agents also varies according to locality. According to **Sharad Shah et al**[21] and **Chrideep et al** from Mumbai, Viral Hepatitis is most common in this scenario, then Malaria and third occupied by Leptospirosis. But WHO bulletin[7] state that Leptospirosis prevalence is more common in South India. Therefore Leptospirosis must occupy the first place in South India. Etiological pattern has to be analysed according to locality.

Leptospirosis is the World Wide Zoonotic infection with much greater incidence in tropical regions. Epidemiology of Leptospirosis has been modified by changes in animal husbandry, climate and human behaviours. The incidence of Leptospirosis is remarkably, underestimated and the disease underdiagnosed in endemic regions. The case fatality rate is 5-25%.

S.K. Mishra et al[23] says that 300 to 400 million cases of Malaria diagnosed yearly and of these 1.5 - 2 million died annually. 90% of world Malarial cases are from tropical Africa and other 10% from 3 countries like India, Sri Lanka and Brazil. Of all the malarial cases in South-East Asia, India constitute 80% Chloroquine-resistant Malaria is in increased prevalence and concept of ACT therapy are now in use.

Early treatment in leptospirosis and Malaria, favour prognosis. Therefore always there will be high degree of suspicion to diagnose these diseases.

WHO, 2004[20] made a statement “more people die of Hepatitis-B in a day than AIDS in an entire year”. National prevalence rate of Hepatitis B was 4.7%, while that of AIDS is less than 1%.

Though Viral hepatitis most of the times need only symptomatic treatment, it is under estimated in our country and now started given importance to this disease.

The study shows etiological analysis of patients present with short duration of fever and jaundice, admitted in Government General Hospital, Chennai and clinical profile of Leptospirosis.

AIMS AND OBJECTIVES

1. To analysis the incidence of various etiological agents for patients get hospitalised in Government General Hospital, Chennai with unexplained fever and jaundice of short duration.

2. To analyse relevant epidemiological data like seasonal variation, incidence, clinical profile, biochemical investigation and incidence of complications on Leptospirosis positive cases.

3. To analyse the immunisation status of Hepatitis B on patients with fever and jaundice.

REVIEW OF LITERATURE

LEPTOSPIROSIS

Severe leptospirosis is otherwise called Weil's disease, Haemorrhagic Jaundice, Mud fever, Swineherd disease, canicola fever, seven-day fever in Japan, cane cutter's disease in Australia, Rice field heptospirosis in Indonesia, Fort Bragg fever in US and Andaman Haemorrhagic fever in Andaman.

HISTORY:

In 1883, Leptospirosis was recognised as an occupational diseases in Sewer workers. In 1886, first description by **Weil,** Professor of Medicine at Heidelberg, of the clinical manifestation seen in men (Severe Jaundice, fever and haemorrhage with renal involvement).

Stimson[33] in 1907, demonstrated by Silver staining, the presence of clumps of spirochetes in the kidney tubules of the patient who reportedly died of yellow fever.

Etiology of Leptospirosis was demonstrated independently in 1915 in Japan and Germany. In Japan, **Inada and Ido**[36] detected both spirochetes and specific antibodies in the blood of Japanese

miners with infectious Jaundice and two groups German Physicians studied german soldiers afflicted by 'French disease' in the trenches of North East France.

Noguchi[36] proposed the name 'Leptospira' in 1918.

TAXONOMY AND CLASSIFICATION:

Serological Classification:

Prior to 1989, the Genus Leptospira was divided into two species, L.Interrogans, comprising all pathological strains and L.biflexa, containing the saprophytic strains.

L. Biflexa was differentiated from L. interrogans by the growth of the former at 13^{o}C and growth in the presence of 8-azaguanine and by the failure of L.biflexa to form special cells in 1M Nacl.

Over 60 serovars of L.biflexa have been recorded. Within the species L-interrogans over 200 serovars are recognised.

GENOTYPIC CLASSIFICATION:

The phenotypic classification of leptospires has been replaced by a genotypic one in which a number of genomespecies include all serovars of both L-interrogans and L.biflexa.

Genospecies of Leptospira and Distribution of Serogroups:

Table - 1

S.No	***Species***	***Serogroups***
1.	L.interrogans	Icterohemorrhagiae, canicola, Pomona, australis, autumnalis, pyrogenes, Grippotyphosa, Djasiman, Hebdomadis etc.
2.	L.noguchi	Panama, autumnalis, pyrogenes, Tarassori, Australis, Shermani, Pomona.
3.	L.santarosai	Shermani, Hebdomadis, pyrogenes, Autumnalis
4.	L.meyeri	Ranarum, Semaranga, Seiroe, Mini
5.	L.wolbachii	Codice
6.	L.biflexa	Semaranga, Andamana
7.	L.fainei	Hurstbridge
8.	L.borgpetersenii	Javanica, Ballum, Hebdomadis, Seiroe, Tarassovi, Mini
9.	L.kirschneri	Grippotyphosa, autumnalis, cynopteri, Hebdomadis etc.

10.	L. Weilii	Celledoni, icterohaemorrhagiae, Sarmin
11.	L.inadai	Lyme, Shermani, icterohaemorrhagiae
12.	L. Parva	Turnesia
13.	L.alexanderi	Manhao, Hebdomadi, Javanica

After studying several hundred strains, workers at centers for Disease Control (CDC) more recently defined 16 genospecies of Leptospira.

Biology of Leptospires:

Leptospire is tightly coiled spirochetes, usually 0.1μm by 6 to 0.1 by 20μm. Two axial filaments with polar insertion are located in the periplasmic space.

Leptospires are obligate aerobes with an optimum growth temperature of 28^0 - 30^oC. It produces both catalase and oxidase.

It grows in simple media enriched with vitamins (Vit B_2 & B_{12} are growth factor), long chain fatty acid, and ammonium salt. Most widely used medium oleic acid - albumin medium EMJH. It contains Tween 80 and bovine serum albumin.

EPIDEMIOLOGY:

Animal Reserviors:

Mammals are most important animal reservoirs. Leptospires are parasites of both wild and domestic animals. Wild variety of animals serve as a source of infection like rats, field mice, hedgehog, fox, mongoose, deer and domestic animals like cattle, sheep, goats and poultry.

In infected animals, initial Leptospiremic phase followed by a period in which organism confined to kidney. Leptospires are excreted in the urine and animal acts as a carrier. Man is accidental host.

It is well known that particular host species may severe as a reservoir for one or more serotypes of Leptospires and conversely a given serotype may be hosted by multiple animal species. Serovars most frequently associated with rodents are icterohemorrhagiae and autumnalis, cattle are pomana and tarassori, sheep and goats are Pomona and grippotyphosa and dogs are canicola and icterohemorrhagiae.

TRANSMISSION TO HUMAN HOST:

Direct– by contact with blood, tissue, organs and urine of infected animals.

Indirect– by exposure to an environment contaminated with Leptospires, water and soil contaminated with infected urine.

Human to Human transmission rare.

Factors favouring survivals of leptospices are moistures, temperature 28-32^{o}C, pH of the soil and surface water 6.2-8. Factors impede the survivals are salinity, chemical pollution and acidic pH.

Flooding after heavy rain is particularly favourable for Leptospirosis. It can survive few hours in dry solid but can survive upto 6 months in flooded conditions. Rats urine contamination of water in wells remains an important mode for the transmission of Leptospires to man.

In Chennai[7], high rainfall and outdoor manual occupation encourages higher incidence rate of Leptospirosis and that more specific source cannot be pinpointed with certainty.

PATHOLOGY AND PATHOGENESIS:

Leptospira penetrate intact mucous membranes and abraded skin and disseminate widely via the blood stream. It produces infectious vasculitis, with damage to capillary endothelial cells responsible for major clinical manifestations of disease including renal tubular and hepatic dysfunction, myocarditis and pulmonary haemorrhage.

In Liver, damage is at subcellular level. An endotoxin like substance in the wall of spirochetes has been suggested. Plasma TNF-α level related to severity of organ involvement. Liver necrosis is minimal and focal. Zone 3 necrosis absent. Active hepatocellular regeneration, is out of proportion to cell damage. Cirrhosis is not a sequalae.

Examination of kidney from autopsy reveals ischemic damage including epithelial cell necrosis in the DCT and ascending loop of henle and interstitial nephritis but only rarely glomerular damage. According to C.W. Yang et al[33], Leptospira outer membrane proteins (OMPs) may elicit tubular injury and inflammation through Toll-like receptor (TLR) dependent pathway followed by activation of nuclear transcription factor Kappa B and mitogen activated protein

kinases and a differential induction of chemokines and cytokines relevant to tubular inflammation.

CLINICAL FEATURES:

Symptoms usually develop 7-12 days after exposure. Clinical presentation of leptospirosis is biphasic with an acute or septicemic phase lasting about a week, followed by the immune phase characterised by antibody production and excretion of leptospires in the urine. Most of the complications occur during immune phase and thus occur during the second week of illness.

ANICTERIC LEPTOSPIROSIS:

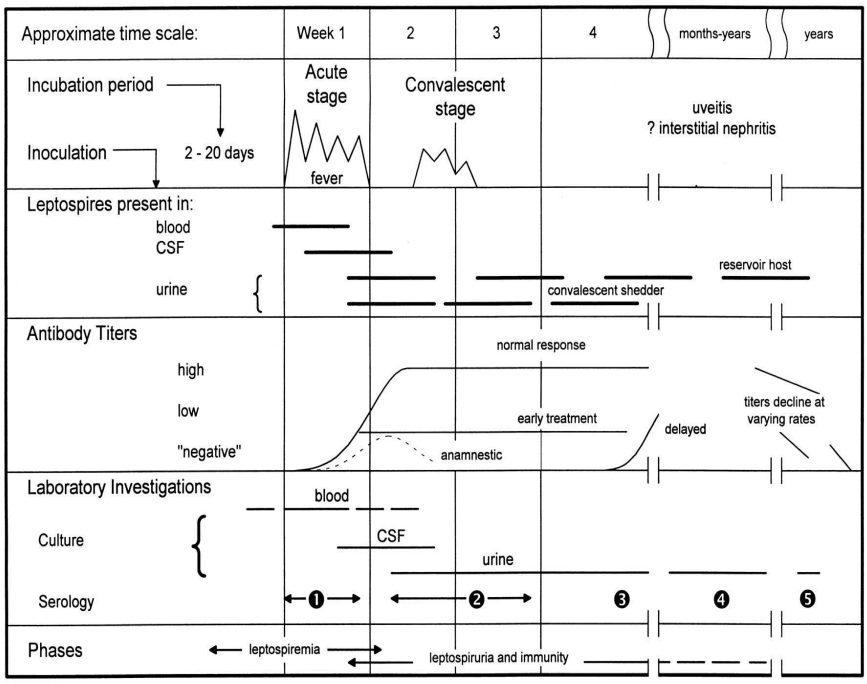
Great majority are subclinical. Patients usually have symptoms like chills, headache, myalgia, abdominal pain and conjunctival suffusion. Myalgia affecting lower back, thighs and calves is often intense. This last few about a week and its resolution coincides with the appearance of antibodies.

ICTERIC LEPTOSPIROSIS:

More severe disease, rapidly progressing. 5-10% of patients of leptospirosis enter into icteric phase. Mortality rate is about 5-15% liver function test return to normal after recovery. Serum bilirubin

may be high, moderate rise of transaminase level and minor elevation of alkaline phosphatase level usually occurs

Figure - 3



Biphasic nature of Leptospirosis and relevant investigations at different stages of disease.

Comparison of symptoms/signs of severe Leptospirosis / Viral Hepatitis:

Table - 2

	Weils Disease	***Viral Hepatitis***
Onset	Sudden	Gradual
Headache	Constant	Occasional

Muscle pain	Severe	Mild
Conjunctival Suffusion	+	–
Disorientation	Common	Rare
Bleeding	+	–
Nausea/Vomiting	+	+
Abdominal Discomfort	+	+
Albuminuria	+	–
Leucocyte Count	Polymorph leucocytosis	Leucopenia with lymphocytosis
CPK	Increased	Normal

DIAGNOSIS:

Microscopic Examination:

Leptospires may be visualised in clinical materials by dark field microscopy or by immunofluorescence or light microscopy after appropriate staining. Approximately 10-Leptospires/ml are necessary for one cell per field to be visible by dark field microscopy.

Dark field microscopy examination of body fluids such as blood, urine, CSF and dialysate fluid has been used, but is both insensitive and lacks specificity.

A variety of histopathological stains have been applied to the detection of Leptospires in tissues. Leptospires were first visualized by Silver staining and the Warthin-Starry stain is widely used for histological examination. Recently, immuno-histochemical methods have been applied.

CULTURE:

Leptospiremia occurs during the first stage of the disease, beginning before the onset of symptoms and ends by first week of the illness. Therefore blood cultures should be taken as soon as possible after the patient presentation.

One or two drops of blood are inoculated into 10ml of semisolid medium containing 5-flurouracil at the patients bedside.

Apart from blood, CSF and dialysate fluid can also be cultured during first week of illness. Urine can be cultured from the beginning of the second week of symptomatic illness.

Cultures are incubated at 28 to 30^{o}C and examined weekly by dark field microscopy for upto 13 weeks before being discarded.

ANTIGEN DETECTION:

Detection of Leptospiral antigens in clinical material offer greater specificity than DFM. Radio immunoassay (RIA) can detect 10^4 to 10^5 sub Leptospires/ml and enzyme-linked immunosorbent assay (ELISA) can detect 10^5 sub Leptospires/ml.

ANTIBODY DETECTION:

Antibodies can become detectable by the 6^{th} to 10^{th} day of disease and generally reach peak levels with 3 to 4 weeks. Antibody level then gradually recede but may remain detectable for years. Serological methods can be divided into two groups: those, which are genus specific and those which are serogroup specific.

Microscopic Agglutination Test (MAT):

The reference method for serological diagnosis of leptospirosis is the MAT, in which patient sera are reacted with live antigen suspensions of Leptospiral serorars. After incubation, the serum antigen mixtures are examined microscopically for agglutination and the titres are determined. The MAT is read by Dark field microscopy. The end point is the highest dilution of serum at which 50% agglutination occur.

Paired sera are required to confirm a diagnosis with certainly. A fourfold or greater rise in titre between paired sera confirms the

diagnosis regardless of the interval between samples. If symptoms of overt Leptospirosis are present, an interval of 3 to 5 days may be adequate to detect rising titres. However, if the patient present earlier in the course of the disease or if the date of onset is not known precisely, then an interval of 10 to 14 days between samples is more appropriate.

In the current CDC case definition[1], a titre of $\geq$200 is used to define a probable case with a clinically compatible illness. In areas where Leptospirosis is endemic, a single titre of $\geq$800 in symptomatic patients is indicative of Leptospirosis. The National Institute of Communicable diseases[2] (NICD), New Delhi case definition also advocates a MAT titre of >200.

Other Serological Tests:

Because of the complexity of the MAT, rapid screening tests for Leptospiral antibodies in acute infection have been developed. Complement fixation (CF) was widely used but methods were not standardized. CF tests have generally been replaced by ELISA method.

IgM detection has repeatedly been shown to be more sensitive than MAT when the first specimen is taken early in the acute phase

of the illness. The drawback of this test is that the infective serovars cannot be assessed. Though this test is more sensitive than MAT, it is less specific.

TREATMENT:

Antibiotic treatment is most beneficial if started within 4 days of illness. Unfortunately, the diagnosis of Leptospirosis is rarely made this rapidly.

Treatment and chemoprophylaxis of Leptospirosis[41]

Table - 3

Treatment of	***Drugs***
Mild Leptospirosis	Doxycycline 100mg orally bd (or) Amoxycillin 500mg orally tid
Moderate / Severe leptospirosis	Penicillin G, 1.5 million units IV qid (or) Ampicillin 1g IV tid (or) Ceftriaxone 1g IV od (or) Cefataxime 1g IV tid
Chemoprophylaxis	Doxycycline 200mg orally once a week

Note: All regimens used for treatment are administered for 7 days.

MALARIA

HISTORY:

Hippocrates called as 1st Malariologist. By 400 BC, he describe various Malarial fever of man. Galen[29], ancient Greek physician noticed first jaundice among malaria infected people. **Charles Louis Alphonsa Laveran**, French physician identified malarial parasite in 1888.

William G. McCallum[29] in 1897, demonstrate sexual process of malarial parasite. **Ronald Ross** identified infected female anaphiline mosquito responsible for transmission.

DIAGNOSIS:

Peripheral smear examination is the corner stone for diagnosis of malaria. Two type of peripheral smear examined thick smear and thin smear. In thick smear, dried for 30 minutes and not fixed with methanol. Allow RBC and leukocytes to haemolyse and any malarial parasite present will be the only detectable element. It is used to detect infection and to estimate parasite concentration.

In thin smear, air dry for 10 minutes, fixed with methanol (dipping the slide into methanol for 5 seconds) used for specification of parasites and sensitivity of thin film is 200 parasites/μl of blood.

QBC Malarial Method:

It was developed by **Becton and Dickenson**[29]. It is positive for Malaria, Babesiosis, Tryponosomiasis, Filariasis and relapsing fever.

METHOD:

QBC tube is a high precision glass hematocrit tube, precoated internally with acridine orange stain and potassium oxalate. QBC tube has to fill with 55 to 65 μl of blood. Tube centrifuged at 12,000 rpm for 5 minutes. Component of buffy coat separated bands. QBC tube is placed on tube holder and examined using a standard white light microscope equipped with the UV microscope adaptor. Fluorescing parasites are then observed at the red blood cell/WBC interface.

Since the parasite contain DNA which take up acridine orange stain, they appear as bright speaks of light among the non-fluorescing red cells. Virtually all the parasite found in 60ml of blood can be visualised by rotating the tube under microscope.

	Peripheral Smear	***QBC Method***
Method	Cumbersome	Easy
Time	Longer 60-120min	Faster 15-30 min
Sensitivity	5 parasite/µl of thick film. 200 parasite/µl of thin film	Claim to be more sensitive at least as good as thick film.
Specificity	Gold standard	? False +ve
Species identification	Gold standard	Not
Cost	Inexpensive	Costly

RAPID DIAGNOSIS OF MALARIA:

Immunochromatographic test for Malarial Antigen:

Based on capture of parasite antigen from the peripheral blood using either monoclonal (or) polyclonal antibodies against the parasite antigen target.

a) Histidine rich protein 2 of plasmodium falciparum

Water soluble protein produced by asexual stage and gametocyte of P.falciparum, expressed on red cell membrane surface. Remains in the blood for 28 days after the initiation of antimalarial therapy.

b) Plasmodium Aldolase:

Enzyme of parasite glycolytic pathway expressed by blood stages of P.falciparum as well as non-falciparum malaria.

c) Parasite lactate dehydrogenases:

Soluble glycolytic enzyme produced by asexual and sexual stages of live parasite found in all 4 species of malaria.

MALARIAL HEPATHOPATHY

Jaundice[23] present in 2.5% of patients with malaria. According to WHO, there is increase number of report favouring existence of malarial hepatopathy from Asian countries especially from India.

Severe haemolysis and rupture of hepatocytes during primary schizogony are responsible for jaundice in malaria. Here there is usually only mild elevation of liver enzymes.

Malarial hepatitis characterised by rise in serum bilirubin along with rise in transaminases more than three times the upper limit of normal.

Histological hallmark of malarial hepatopathy is presence of acute injury to hepatocytes and deposition of malarial pigment.

Study conducted by **Murthy et al**[24] shows 21% of patients with falciparum malaria has malarial hepatitis.

ACUTE VIRAL HEPATITIS

Acute viral hepatitis is characterised by constitutional symptoms like anorexia, nausea/vomiting, arthralgia, myalgia, headache, cough etc., may precede the onset of jaundice by 1-2 weeks with the onset of jaundice, the constitutional prodromal symptoms usually diminish. Liver become enlarged and tender. Splenomegaly and lymphadenopathy present in 10-15% of patients.

During the recovery phase, constitutional symptoms disappear. Some liver enlargement and abnormal biochemical values still persist. Duration of post icteric phase variable ranging from 2-12 wks.

Serum aminotransferase increase during the prodromal period itself and precede the rise in bilirubin level. Prolonged prothrombin time and low albumin level in viral hepatitis indicate worse prognosis.

All acute viral hepatitis needs only supportive therapy except Hepatitis C which need peglylated interferon α and Ribavarin for 48 weeks.

TYPHOID HEPATITIS

Hepatitic complications of typhoid fever first reported by **William Osler**[31] in 1899. Salmonella organisms[31] are phagocytosed by reticuloendothelial system by overcome the cell's killing action and produce hepatic injury and liberate toxin.

Liver biopsy shows focal Kupffer cell hyperplasia and mononuclear cell infiltration of the portal space.

MATERIALS AND METHODS

SELECTION CRITERIA:

- Patients with acute onset of fever and jaundice of less than 20 days duration were included in the study.
- All patients were >12 years of age.

EXCLUSION CRITERIA:

- Patients with chronic liver disease were excluded from this study.
- All pediatric cases were excluded from this study.
- All retroviral diseases were excluded from this study.

No. of patients studied: 142.

Period of study: Between January 2008 to October 2008.

Study Design: Prospective Study

Ethical Clearance: Obtained.

METHODS:

All patients admitted in Govt. General Hospital, Chennai with fever and jaundice were evaluated. Consent for study obtained. Detailed history was taken regarding duration of fever and jaundice, occupation and immunisation against Hepatitis A and Hepatitis B viruses. Thorough clinical examination was done in all cases.

Documentation was done using a stratified proforma which included symptoms like Jaundice, Fever, Headache, Myalgia, vomiting, chills & rigor, conjunctival suffusion, Bleeding manifestation, abdominal pain, diarrhea, oliguria, cough & burning micturitun.

Signs like icterus, rashes, signs of dehydration, anaemia, splenomegaly, hepatomegaly, lymphadenopathy, muscle tenderness, pedal edema, altered sensorium, and signs of meningeal irritations were noted.

Investigations like complete haemogram, renal function test and liver function test on every 3rd day, urine routine, 12 lead ECG, x-ray chest, ultrasonogram of abdomen, smear for Malarial parasite, widal test, macroscopic slide agglutination test (MSAT) and

microscopic agglutination test (MAT) for Leptospirosis, viral markers like IgM Anti-HAV antibody, HbsAg and anti HCV, and blood-culture and sensitivity were done on all patients.

If the patients has acute renal failure, renal function test was done daily.

Follow up of all patients regarding treatment and outcome were done during the hospital stay.

Eye manifestations of Leptospirosis done with detailed history and fundus examination.

RESULTS

Number of cases admitted with fever and jaundice: 142

Number of male cases : 91 (64%)

Number of female cases : 51 (36%)

Age and Sex Distribution:

Table 4

Age	***Number of patients***	
	Male (%)	***Female (%)***
12-20 years	11 (7.7%)	5 (3.5%)
21-30 years	17 (11.9%)	13 (9.1%)
31-40 years	31 (21.8%)	16 (11.2%)
41-50 years	19 (13.3%)	9 (6.3%)
>50 years	13 (9.1%)	8 (5.6%)

Etiological Agent:

Table 5

Agent	***Number of patients n=142***	***Percentage***
Leptospirosis	50	35.2%
Malaria	24	16.9%
Viral Hepatitis	19	13.3%
Gall bladder disease	3	2.1%
Unknown	46	32.3%

ANALYSIS OF LEPTOSPIROSIS POSITIVE CASES

A Leptospirosis positive case is one with a MAT titre of more than 1:200 among patients presenting with fever and jaundice - as per CDC[1] case definition 1997 and National Institute of Communicable Diseases[2] - NICD case definition 2001.

Number of cases found serologically positive for leptospirosis: 50.

One patient positive for both Leptospirosis and Hepatitis A.

Age Distribution:

Table 6

Age	***Number of patients n=50***	***Percentage***
12-20 years	9	18%
21-30 years	9	18%
31-40 years	15	30%
41-50 years	10	20%
>50 years	7	14%

Sex Distribution:

Number of male cases : 33 (66%)

Number of female cases : 17 (34%)

Distribution of signs and symptoms:

Table 6

Symptoms/Signs	***Number of patients n=50***	***Percentage***
Fever	50	100%
Jaundice	50	100%
Headache	31	62%
Myalgia	47	94%
Chills/rigor	13	26%
Nausea/Vomiting	20	40%
Cough	18	36%
Conjunctival Suffusion	38	76%
Abdominal pain	10	20%
Diarrhea	12	24%
Oliguria	8	16%
Skin rash	2	4%
Dehydration	12	24%
Pallor	13	26%
Pedal Edema	10	20%
Lymphadenopathy	1	2%
Hepatomegaly	10	20%
Splenomegaly	4	8%
Muscle tenderness	20	40%
Altered sensorium	15	30%
Meningeal sign	0	0%

Average duration of fever on presentation is 9.6 days.

Average duration of jaundice on presentation is 4.8 days.

Ocular manifestations:

Table 7

Signs	***Number of patients n=50***	***Percentage***
Conjunctival Hyperemia	38	76%
Subconjunctival Haemorrhage	3	6%
Fundus abnormality	0	0%

Occupation:

Table 8

Occupation	***Number of patients n=50***	***Percentage***
Manual workers	30	60%
Non-Manual workers	8	16%
Students	4	8%
Others	8	16%

ANALYSIS OF LABORATORY INVESTIGATIONS OF LEPTOSPORISIS CASES

COMPLETE HEMOGRAM:

Hemoglobin:

- The mean Hemoglobin values was 10.5gm%.

- Range 7.1 - 14.0 gm%
- 3 patients had Hb% <8 gm%

WBC Count:

- The mean total WBC count was 6500 cells/µl
- Range 4100-12000 cells/µl.

Platelet Count:

- The mean platelet count was 1.67 lakhs/µl.
- Range 31,000-3,33,000/µl.
- Six patients had platelet count <1 lakh/µl.

Erythrocyte sedimentation rate:

- The mean ESR at 1 hr was 26mm.
- Range 6-120mm.

LIVER FUNCTION TESTS:

Total bilirubin:

Table 9

Total Bilirubin (mg/dl)	***Number of patients n=50***	***Percentage***
2.1 – 5	19	38%
5.1 – 10	17	34%
10.1 – 15	11	22%
>15	3	6%

Table 10

On Admission	*mean*	*Range*
Total bilirubin	7.8mg/dl	2.1 - 38
Indirect bilirubin	3.3mg/dl	0.4 - 10.1
Direct bilirubin	4.7mg/dl	1.1 - 28
SGOT (U/L)	74.6 U/L	35-240
SGPT (U/L)	75.08 U/L	26-300
Alkaline phosphatase (gm/dl)	133.14g/dl	64-240

Renal function test:

Table 11

Test	*Mean*	*Range*
Blood Urea (mg/dl)	56.72	24-180
Sr. Creatinine (mg/dl)	1.73	0.6-8

Blood Urea levels:

Table 12

Urea (mg/dl)	*Number of patients n=50*	*Percentage*
<40	30	60%
40-80	11	22%
80-120	5	10%
>120	4	8%

Sr. Creatinine Levels:

Table 13

Sr.Creatinine (mg/dl)	***Number of patients n=50***	***Percentage***
<1.2	31	62%
1.2 - 3.0	10	20%
3.0 - 5.0	6	12%
>5.0	3	6%

Serovars Analysis:

Table 14

Serovars	***Number of patients n=50***	***Percentage***
Icterohemorrhagiae	20	40%
Semaranga	16	32%
Grippotyphosa	6	12%
Automnalis	5	10%
Australis	3	6%

Serovars and Complication correlations:

Table 15

Serovars	***Number of patients n=21 (%)***	***Renal failure n=19 (%)***
Icterohemorrhagiae	10 (47%)	14 (74%)
Semaranga	4 (19%)	–
Grippotyphosa	3 (14%)	2 (10.5%)
Automnalis	3 (14%)	2 (10.5%)
Australis	1 (4%)	1 (5%)

After rehydration, renal function test returns to normal in 10 patients.

Outcome:

Total number of Leptospirosis positive cases: 50

Number of cases expired : 5

Mortality rate : 10%

ANALYSIS OF MALARIA POSITIVE CASES

Number of cases positive for malaria in patients presented with fever and jaundice : 24

Number of Plasmodium falciparum cases : 17

Number of Plasmodium vivax cases : 7

SYMPTOMS AND SIGNS OF MALARIAL CASES:

Table 16

Symptoms/Signs	***Number of patients n=24***	***Percentage***
Headache	21	87.5%
Chills/rigor	22	91.6%
Nausea/Vomiting	11	45.8%
Myalgia	4	16.7%
Conjunctival Suffusion	2	8.3%
Oliguria	4	16.7%

Symptoms/Signs	*Number of patients n=24*	*Percentage*
Pallor	12	50%
Splenomegaly	9	37.5%
Altered sensorium	10	41.7%

Complication of malaria:

Table 17

Complications	*Number of patients n=24*	*Percentage*
Anaemia	16	66.7%
Thrombocytopenia	12	50%
Malarial Hepatopathy	4	16.7%
Renal Failure	7	29.2%

Hepatitis virus marker pattern:

Table 18

Hepatitis Virus	*Number of patients n=19*	*Percentage*
Hepatitis A	4	21%
Hepatitis B	10	52.6%
Hepatitis C	5	26.3%

DISCUSSION

Among the patients present with history of short duration of fever and jaundice, in our study Leptospirosis constitute about 16.9% Malaria 16.9% and Viral hepatitis 13.3%.

Study conducted in Thanjavur Medical College[8] in 2006, which was non-endemic area for Leptospirosis, shows Leptospiral positivity rate of 24%. Also study conducted in puduchery[8] shows Leptospirosis in 12% of cases of fever with jaundice.

Our study shows Leptospirosis in .35.2%. Therefore Leptospirosis is most common cause of patients present with short duration of fever and jaundice in chennai and the incidence of Leptospirosis also very high among these patients when compared to other studies.

Study conducted in Department of Medicine, Lady Hardinge Medical College Hospital, New Delhi in 2000, among patients presented with acute liver failure[28] (fever and jaundice) shows Viral hepatitis 52.7%, Malaria 19.4% and Leptospirosis 16.7%.

Also study conducted by Department of Paediatric, KEM hospital[35], Mumbai, shows Viral hepatitis 61%, Another study

conducted in Rajahmundry town of Andhrapradesh[20] during 1997-1998 during jaundice epidemic shows Viral hepatitis 45.8%.

WHO bulletin, 2000[12] shows that in India, Seropositivity rate for Leptospirosis vary from place to place.

South India : 25.6%

North India : 8.3%

West India : 3.5%

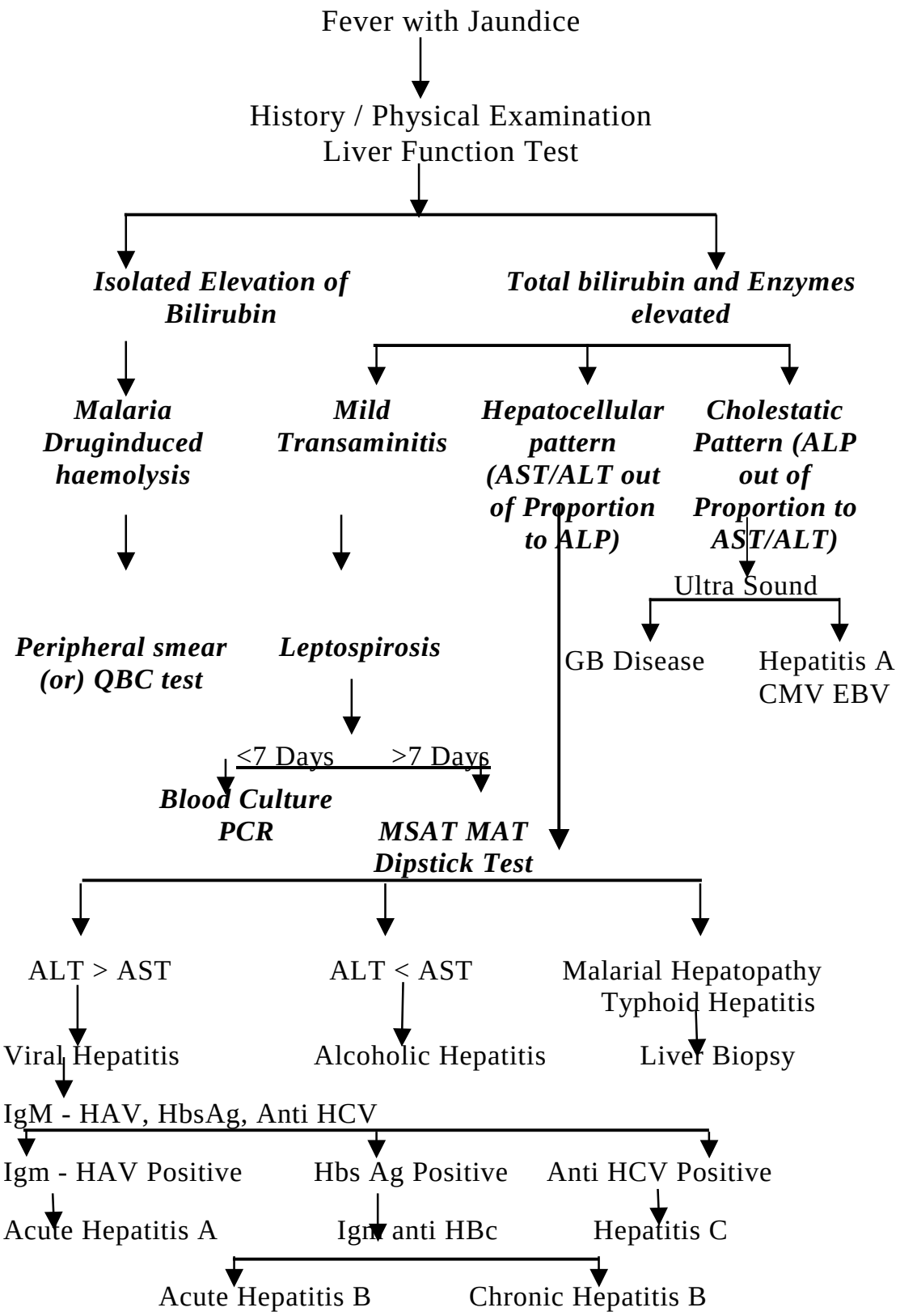
East India : 3.1%

This clearly shows that Leptospirosis is more prevalent in South India.

All studies[20,21,26] conducted in North India shows that viral hepatitis constitute major etiology among patients presented with fever and Jaundice. But in our study, Leptospirosis constitute major etiology. This change of pattern is due to high seroprevalence of Leptospirosis in South India.

Also algorithm for management of short duration of fever and jaundice has to programmed according to local prevalence of etiological agent and age pattern.

ALGORITHM


Fever with Jaundice
History / Physical Examination
Liver Function Test
Isolated Elevation of Bilirubin
Total bilirubin and Enzymes elevated
Malaria Druginduced haemolysis
Mild Transaminitis
Hepatocellular pattern (AST/ALT out of Proportion to ALP)
Cholestatic Pattern (ALP out of Proportion to AST/ALT)
Ultra Sound
Peripheral smear (or) QBC test
Leptospirosis
GB Disease
Hepatitis A
CMV EBV
<7 Days
>7 Days
Blood Culture
PCR
MSAT MAT
Dipstick Test
ALT > AST
ALT < AST
Malarial Hepatopathy
Typhoid Hepatitis
Viral Hepatitis
Alcoholic Hepatitis
Liver Biopsy
IgM - HAV, HbsAg, Anti HCV
Igm - HAV Positive
Hbs Ag Positive
Anti HCV Positive
Acute Hepatitis A
Igm anti HBc
Hepatitis C
Acute Hepatitis B
Chronic Hepatitis B

ANALYSIS OF LEPTOSPIROSIS POSITIVE CASES

Of 142 patients analysed for short duration fever and jaundice, 50 patients have Leptospirosis positive.

Males are predominantly affected due to outdoor work in the field. Mostly 30-40 years of age is predominantly affected.

Manual workers in the field occupied maximum of 60%.

COMPARING OCCUPATION PATTERN

Table 19

Occupation	*GGH, Chennai 1990[8] n=57*	*Present Study n=50*
Manual Worker	57.8%	60%
Non-Manual worker	22.8%	16%
Others	19.4%	24%

COMPARING MONTHLY INCIDENCE OF LEPTOSPIROSIS

Table 20

	Jan	*Feb*	*March*	*July*	*Sep*	*Oct*	*Nov*
GGH, Chennai[8] 1987-93	5	1	–	1	4	65	100
Present Study	1	–	4	3	16	10	–

Both studies show sharp raise in incidence of Leptospirosis after monsoon months like September and October.

Symptoms that are more common for Leptospirosis like Myalgia and Conjunctival suffusion constitute 92% and 76% respectively.

Other symptoms found in high percentages are headache 62%, Nausea/vomiting 40%, cough 36%, abdominal pain 20%, Diarrhea 24% and Oliguria 16%.

Sign analysis shows muscle tenderness in 40%, Altered sensorium 30%, pallor 26%, Pedal edema 20%, Hepatomegaly 20% and splenomegaly 8%.

Comparison of sings and symptoms on admission in patients with Leptospirosis in large series and in this study:

Table 21

Signs and symptoms	***China[36] 1965 n=168***	***India[36] 1992 n=57***	***Brazil[36] 1999 n=193***	***Present study n=50***
Jaundice	0	84	95	100
Headache	90	26	92	62
Conjunctival suffusion	57	58	35	76

Signs and symptoms	***China[36] 1965 n=168***	***India[36] 1992 n=57***	***Brazil[36] 1999 n=193***	***Present study n=50***
Vomiting	18	58	–	40
Myalgia	64	82	94	94
Abdominal pain	26	18	–	20
Dehydration	–	39	–	24
Cough	57	32	–	36
Diarrhea	20	26	–	24
Hepatomegaly	28	–	–	20
Lymphadenopathy	49	–	–	2
Skin rash	–	–	–	4
Meningeal sign	–	7	–	0
Altered sensorium	–	42	–	30

In ocular manifestation of Leptospirosis, conjunctival hyperemia in 76% sub conjunctival haemorrhage 6% and no fundus abnormality. All patients with subconjunctival haemorrhage have low platelet. One patient also positive for IgM - Dengue antibody.

Ocular manifestation in comparison with large series:

Table 22

	MG. Martin[25] et al	***Hetal[25] et al***	***Present study***
Conjunctival Hyperemia	86%	76%	76%
Subconjunctival Haemorrhage	19%	25%	6%
Fundus changes	48%	15%	0%

Our study has low incidence of subconjunctival haemorrhage and fundus changes. This is because of change in pattern of serovars. Fundus changes more commonly documented on these studies was retinal haemorrhage. In our study, over all bleeding manifestation is reduced compared to other large series studies.

ANALYSIS OF LABORATORY INVESTIGATIONS

Complete Haemogram:

Complete haemogram revealed that most patients had haemoglobin level more than 10gm%. Only three patients had haemoglobin level less than 8gm%. Anaemia may be due to obscured bleeding.

The total count of WBC revealed mild leucocytosis in three cases.

Most of them had normal platelet count, except for six patients who had platelet count less than 1 Lkh/μn. Most of these patients had bleeding manifestations.

There was a mild rise in erythrocyte sedimentation rate in most of the patients. One of the patients had ESR above 100mm in 1 hour.

It is evident from these observations that non-specific changes in haemogram can occur in leptospirosis. Though they are not useful in the diagnosis of disease, they are helpful in suspicion of the disease.

LIVER FUNCTION TEST:

The analysis of LFT reveals a high level of bilirubin on admission ranging from 2.1 to 38 mg/dl. There was a conjugated hyperbilirubinemia in most of the cases.

There was mild increase in serum transaminase level while there was a moderate increase in serum alkaline phosphatase levels. The total protein level was within normal limits. Further follow-up of the patients during the hospital stay revealed that the enzyme levels returned to normal on treatment.

RENAL FUNCTION TEST:

While only eight patients presented with oliguria, laboratory investigations revealed mild to severe elevation of blood urea and serum Creatinine level in 38% of patients. After hydration, in ten patients, renal function returned to normal. Nine patients taken for haemodialysis by nephrologists and three of them expired.

Biochemical values compared with study conducted in Chennai.

Table 23

Parameters	***Abnormal Values***	***Muthusethupathi[7] et al n=57***	***Present Study n=50***
Bl. Urea	>40mg/dl	72%	40%
Sr. Creatinine	>1.2mmg/dl	72%	38%
Total bilirubin	>2mg/dl	82%	100%
SGOT	>40mg/dl	44%	78%
SGPT	>40mg/dl	47%	78%
Platelets	>1Lakh/µl	23%	12%

Muthusethupathi[7] et al study include only fever cases positive for Leptospirosis but present study patients present with fever and jaundice, positive for Leptospirosis. Therefore total bilirubin and liver enzymes are in high level compared to previous study. Over all complication rate of Leptospirosis is in decline.

Most common serovars in our study was Icterohaemorrhagiae (40%), semaranga 32%, Grippotyphosa 12% etc.

Serovars Semarange belongs to non-pathogenic serovars and in our study it occupied 32%. This may be because in our microbiology laboratory, they named unidentified species as serovar semaranga. This may be reason why semaranga serovars produce complications in our study.

Study conducted in Andaman island by **Sameer Sharma et al**[39] about serovars prevalence among high risk group shows serovars Grippotyphosa and Australis as Commonest serovars.

Another study conducted near Madras city following an outbreak of the disease in cattle shows automnalis antigen 62% **(Ratnam et al**[40]**)**

Complications like thrombocytopenia and renal failure is more common with icterohaemorrhagea in Chennai.

All the patients diagnosed to have Leptospirosis started on intravenous Benzyl Penicillin for a period of 10 days.

This study also reveal a mortality rate of complicated icteric Leptospirosis as 10%. **Levett PN**[36] gives the mortality rate as 5-15%.

Comparative analysis of leptospirosis/Malaria/Viral Hepatitis:

Table 24

Disease	*Incidence*	*Common Symptoms*	*Liver function test pattern*	*Complication*	*Mortality Rate*
Leptospirosis	35.2%	Myalgia (94%) Conjunctival suffusion (76%)	Conjugated hyperbilirubinemia Minimal transaminase elevation	Thrombocytopenia (42%) Renal failure (38%)	10%
Malaria	16.9%	Chills/ Rigor (92%) Headache (87%)	Unconjugated hyperbilirubinemia Mild elevation of liver enzymes	Anaemia (66.9%) Thrombo-cytopenia (50%)	8%
Viral hepatitis	13.3%	Nausea vomiting (79%) Myalgia (47%)	Conjugated hyperbilirubinemia Very high enzymes level AST > ALT	Anaemia (57.8%)	5%

ANALYSIS OF MALARIA POSITIVE CASES

Among the 142 cases, 24 cases are positive for malarial parasite. Of 24 cases, 17 cases positive for falciparum malaria and 7 cases positive for vivax malaria. Though vivax malaria is more prevalent that falciparum malaria in India, in our study falciparum malaria is common because we include only complicated malaria (fever with jaundice). Complicated malaria is more common with falciparum malaria.

Symptoms more common in complicated malaria cases like Headache and chills/rigor present in 87.5% and 91.6% respectively.

Compared to other series:

Table 25

Symptoms	***Murthy[24] et al***	***Echeverri M[30] et al***	***Present Study***
Chills/Rigor	98.1%	91%	91.6%
Altered Sensorium	48.1%	–	47.1%
Headache	–	91%	87.5%
Oliguria	7%	–	16.7%
Anaemia	75%	46%	66.7%
Thrombo cytopenia	40%	8%	50%
Renal failure	25%	–	29.2%

Murthy et al[24] study include mostly falciparum malaria like our study but Echeverri M et al[30] study conducted in Columbia and include mostly vivax malaria.

Also study conducted by **Sivakumar et al**[6] shows malarial ARF 10%, Leptospiral ARF 45%.

In our study, Malarial hepatopathy documented in 16.7%. In **Murthy et al**[24] study, Malarial hepatopathy constitute 21%.

In this study among the viral hepatitis[19] cases, 21% due to Hepatitis A[4] ,52.6% due to hepatitis B[10] and 26.3% due to hepatitis C. Viral hepatitis pattern in Rajamundry town of Andhrapradesh[28], Hepatitis A 13%, Hepatitis B 28%, Hepatitis C 2.1%, Hepatitis D 13%, and Hepatitis E 42.1%.

In other series

Table 26

Viral Markers	***Khanna et al[20]***	***R.Kaur et al[21]***	***Present study***
Anti IgM HAV	9%	2.3%	21%
Hbs Ag	16%	12.7%	52.6%
Anti HCV	2%	3.3%	26.3%
Anti HEV	67%	20.6%	–

Analysis of viral markers pattern in North India [20,21,27] shows that hepatitis E prevalence in on increase. One of the important limitations of our study is we have not done Ig M anti HEV, due to non availability in our institute. Therefore viral marker pattern can not be compared with longer series due to lack of Ig M anti HEV.

Of the 142 patients included in our study only one patient was immunised against hepatitis B. In 1991, WHO ordered inclusion of Hepatitis B vaccine in National immunization program in high endemic countries by 1995 and in all countries by 1997. In 2001, WHO set new goal of 90% coverage with 3 doses regimen of Hepatitis B Vaccine for all children by 2015.

LIMITATIONS OF THIS STUDY:

1) Since we have small sample size, individual disease and parameter comparison with large series may produce Type II error.

2) In our microbiology laboratory, they named every unidentified serovars as semaranga. This will falsely report more semaranga serovars.

3) We haven’t done IgM - anti HEV for diagnosis of Hepatitis E. due to non availability in our institute. So we can’t compared with large series Hepatitis viral marker pattern.

4) We have not demonstrated four fold rise in MAT titre for diagnosis of Leptospirosis due to limited resource in our institution.

CONCLUSION

1) Among patients present with short duration of fever and jaundice, have etiological agents, in decreasing order of frequency as Leptospirosis 35.2%, Malaria 16.9% and Viral hepatitis 13.3%.

2) Most of the cases of Leptospirosis are from North Madras, during the monsoon months like September, October and among manual workers (60%).

3) Apart from fever and jaundice, the most common symptoms/signs in patients with Leptospirosis are Myalgia, Headache and conjunctival suffusion. Malaria are chills/rigor, Headache and viral hepatitis are nausea/ vomiting or myalgia.

4) The Leptospirosis and Viral hepatitis cases have conjugated hyperbilirubinemia whereas malarial cases have unconjugated hyperbilirubinemia.

5) All patients positive for Leptospirosis responded well to intravenous Benzyl penicillin.

6) Nine cases went into intrinsic acute renal failure, taken for haemodialysis and three of them expired.

7) Most common serovars in our study was icterohaemorrhagea.

8) Virulance of icterohaemorrhagea is on decreasing trend, compared to previous study.

9) The over all mortality of icteric Leptospirosis cases was 10%, Malarial case 8% and Viral hepatitis 5%.

10) Malarial hepatopathy and renal failure associated with 16.7% and 29.2% of malaria positive cases.

11) Among the acute viral hepatitis, Hepatitis B constitute most common causes among adults.

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KEY TO MASTER CHART

NMW	-	Non Manual Worker
MW	-	Manual Worker
S	-	Student
O	-	Others (Retired, House wife etc.,)
N	-	Normal
PV	-	Plasmodium Vivax Malaria
PF	-	Plasmodium Falciparum Malaria
Ictero	-	Icterohaemorrahagea
+	-	Positive
-	-	Negative

INSTITUTIONAL ETHICAL COMMITTEE
GOVERNMENT GENERAL HOSPITAL & MADRAS MEDICAL COLLEGE,
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K.Dis.No.16328 P & D3/Ethies/Dean/GGH/08 Dated 8/9/2008

Title of the work	:	"A Study on the Analysis of Etiology for patient present with fever and Jaundice and to Analyse clinical Profile of those who have leptospirosis
Principal Investigator	:	Dr. J. Bino John Sahayo
Department	:	General Medicine, MMC, Chennai - 3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 10th Sep 2008 at 2 P.M in GGH , Deans, Chamber, Chennai-3.

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate form the area of the work for which I applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s)
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, GGH,CHENNAI

CHAIRMAN
IEC, GGH,CHENNAI

DEAN
GGH &MMC, CHENNAI

RKM.5.6(2)

PROFORMA

Name : ***Ip. No :***

Age : ***DOA :***

Sex : ***DOD :***

Address :

Occupation : Manual / Non Manual worker / Other

Immunisation :

Symptoms : (At admission)

Jaundice : ..... days ***Conjunctival suffusion :***

Fever : .....days ***Bleeding manifestation :***

Head Ache :

Myalgia : ***Abdominal pain :***

Chills & Rigor : ***Diarrhea :***

Nausea / vomiting : ***Oliguria :***

Cough : ***Burning Micturition :***

Others :

Signs : (At admission)

Skin Rash : ***Splenomegaly :***

Hydration : ***Muscle Tenderness :***

Pallor : ***Altered sensorium :***

Pedal edema : ***Meningeal signs :***

Lymphadenopathy : ***Fundus examination:***

Hepatomegaly :

Investigation :

1. **Haemogram** : ***Hb :.....gm %***

TC :.....Cells/u

DC : P.....L.....E.....

Platelet Count :.....

RBC :.....Cells /Ul

ESR ½ hr :.....mm

1 hr :.....mm

2.

Test	***Day 1***	***Rpt.(.....day)***	***Rpt (.....day)***
Bl. Sugar (mg/dl)			
Bl. Urea (mg/dl)			
Sr. Creatinine(mEq/l)			
Sr. Na+(mEq/l)			
Sr . K+(mEq/l)			

3. Urine : Albumin :

Sugar :

Deposit :

Bile salt :

Bile pigment :

4. Liver Function Test :

Test	*Day 1*	*Rpt (.....day)*	*Rpt (.....day)*
Total bilirubin (mg/ dl)			
Direct Bilirubin(mg/dl)			
Indirect bilirubin(mg/dl)			
SGOT (U/L)			
SGPT (U/L)			
Sr. Alkaline phosphatise(Iu/l)			
Total protein (g/dl)			

5. X- ray Chest PA View :

6. ECG :

7. USG . abd & pelvis :

8. Mp/Mf :

9. WIDAL :

10. Blood Culture & sensitivity :

11. MSAT :

12. MAT :

13.IgM anti HAV

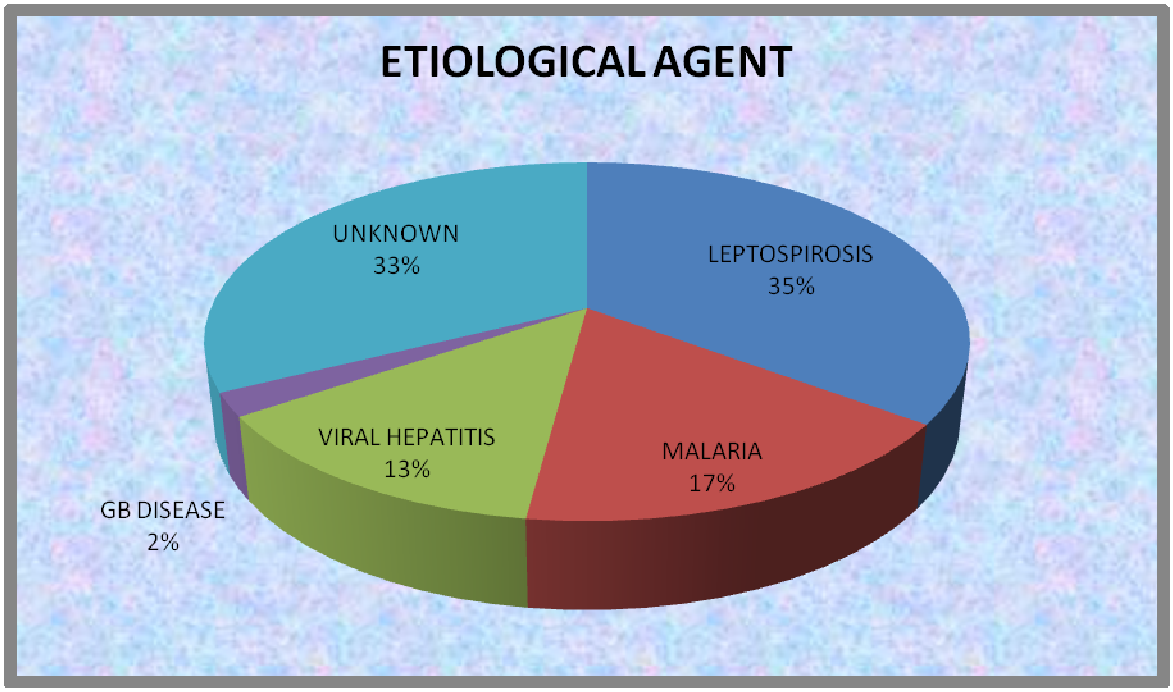
14. Sr. HbsAg:

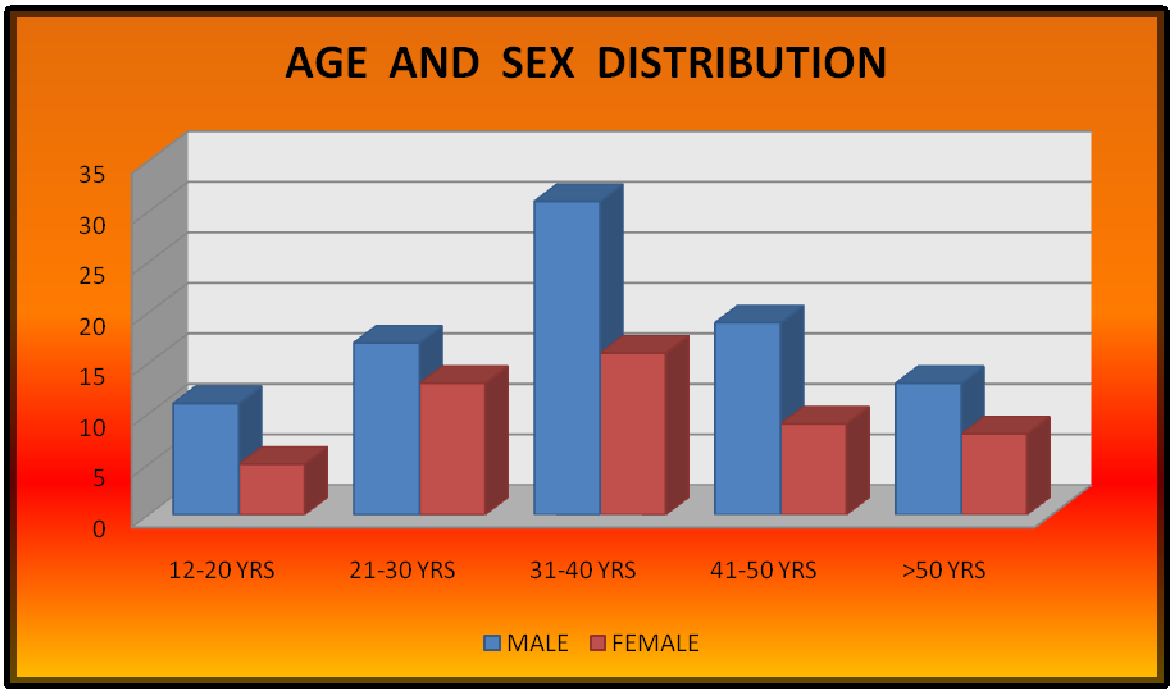
15.Anti-HCV

Treatment given :

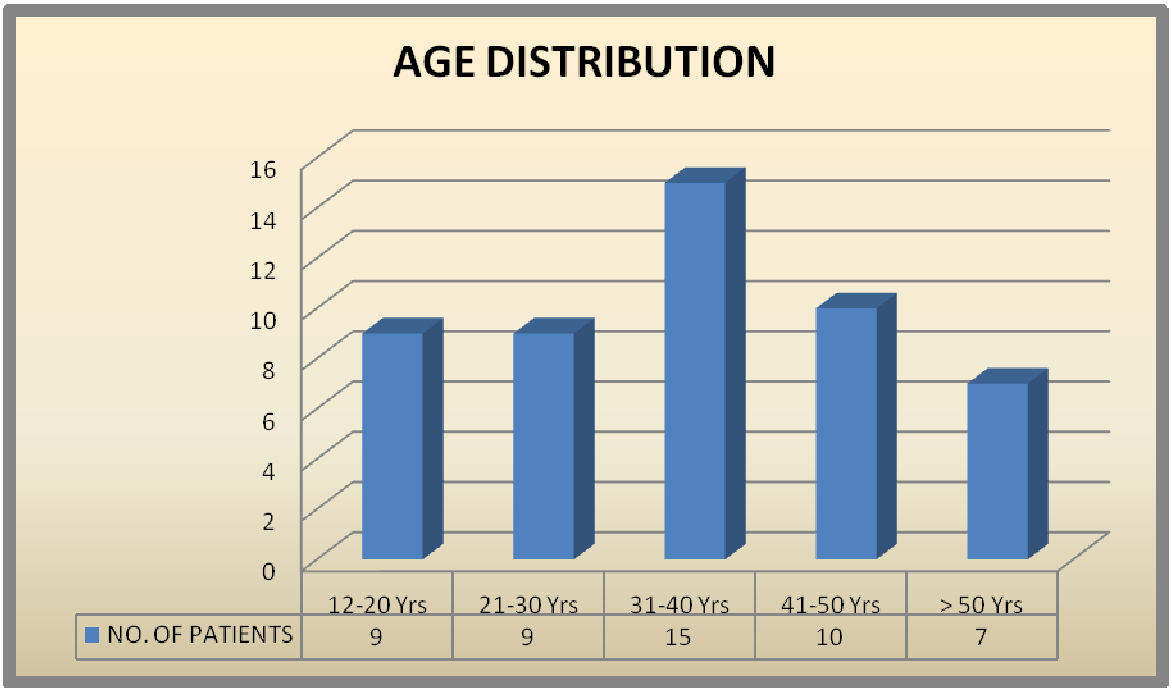
Outcome

○ ***Cured*** ○ ***Death***

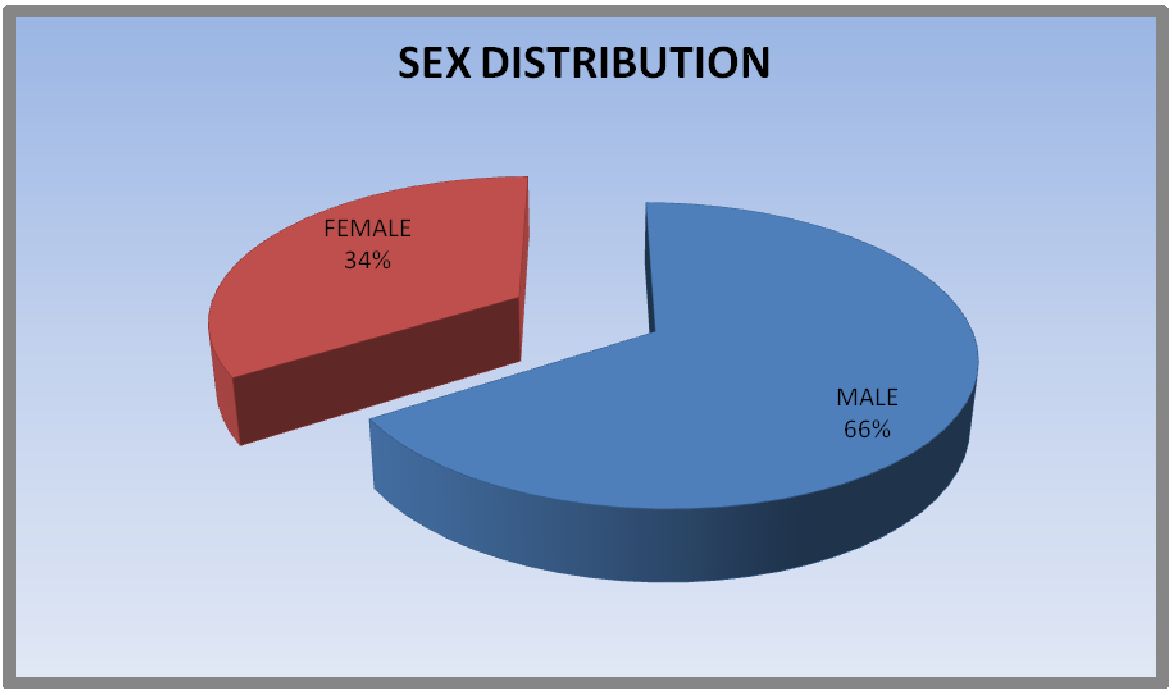

ETIOLOGICAL AGENT
UNKNOWN
33%
LEPTOSPIROSIS
35%
VIRAL HEPATITIS
13%
MALARIA
17%
GB DISEASE
2%

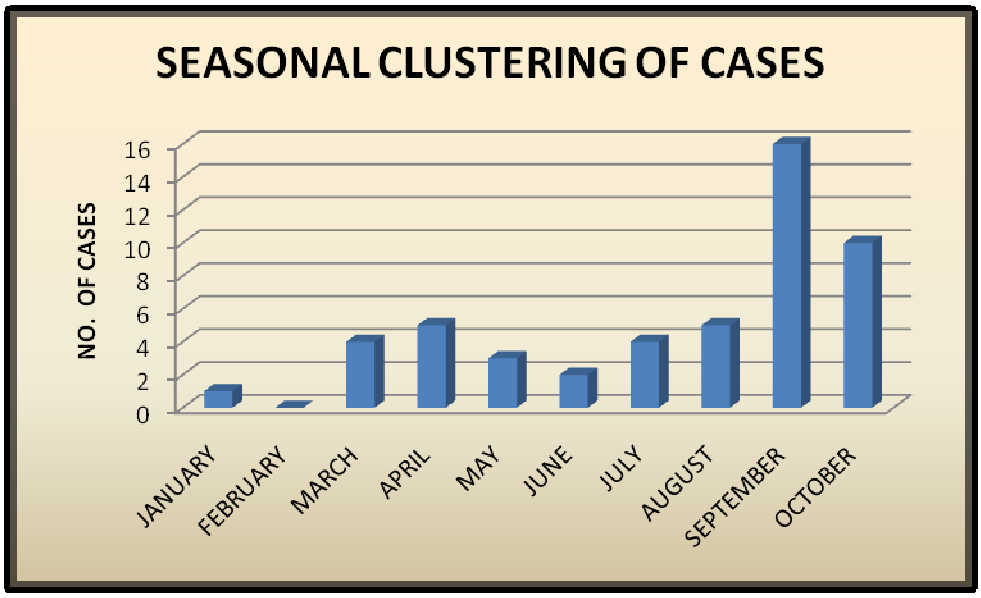

AGE AND SEX DISTRIBUTION
35
30
25
20
15
10
5
0
12-20 YRS
21-30 YRS
31-40 YRS
41-50 YRS
>50 YRS
MALE
FEMALE

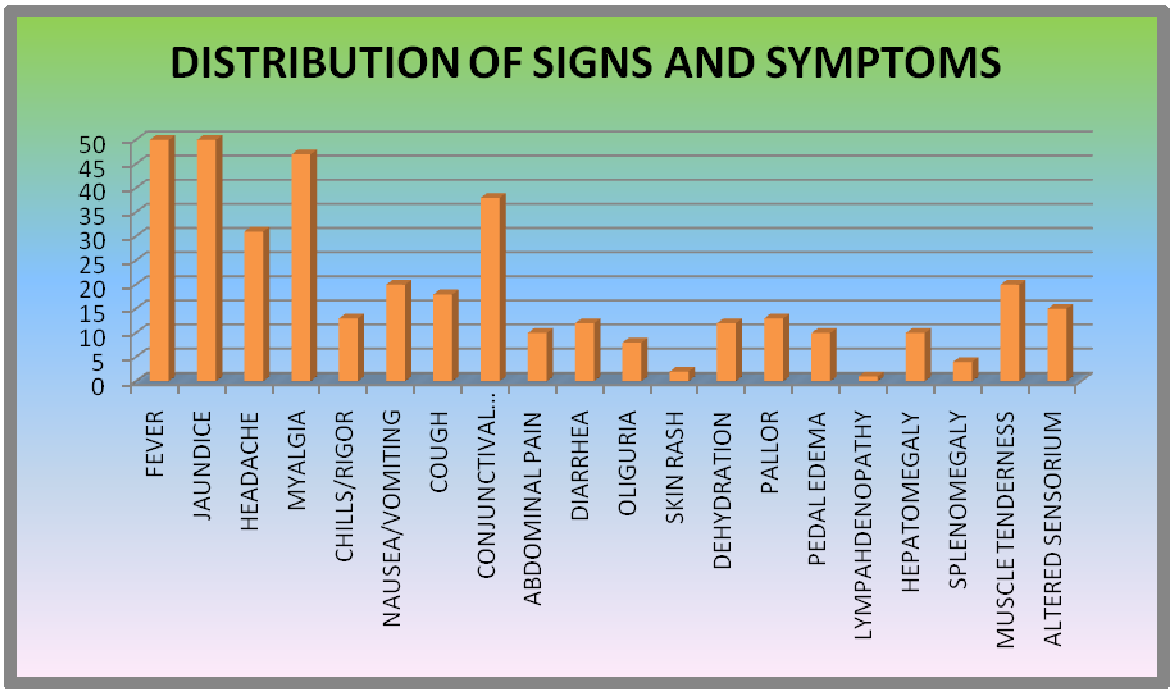
AGE DISTRIBUTION

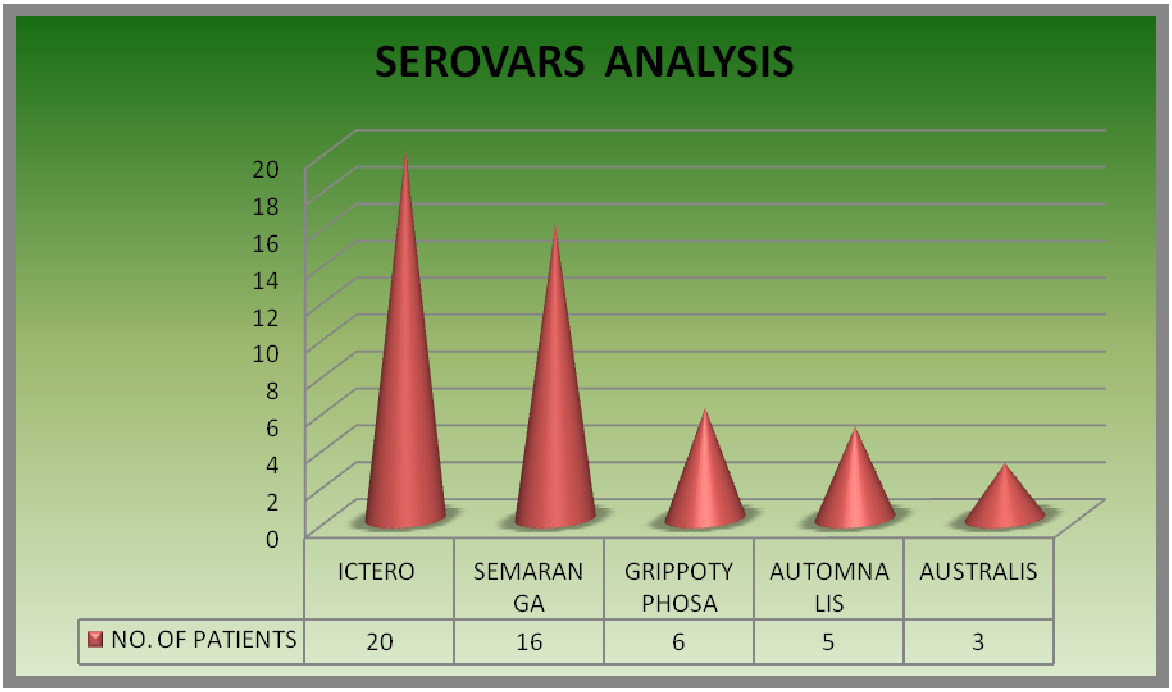


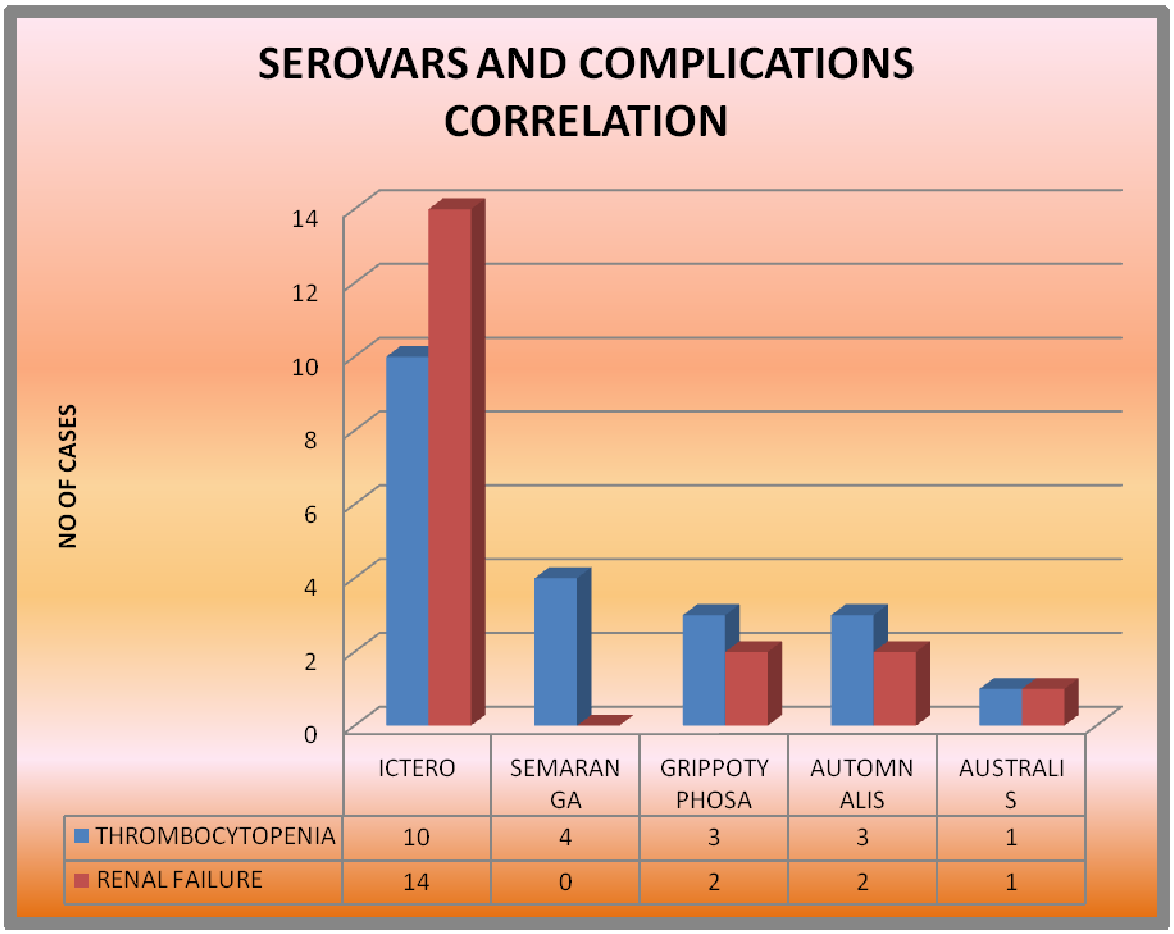
SEX DISTRIBUTION

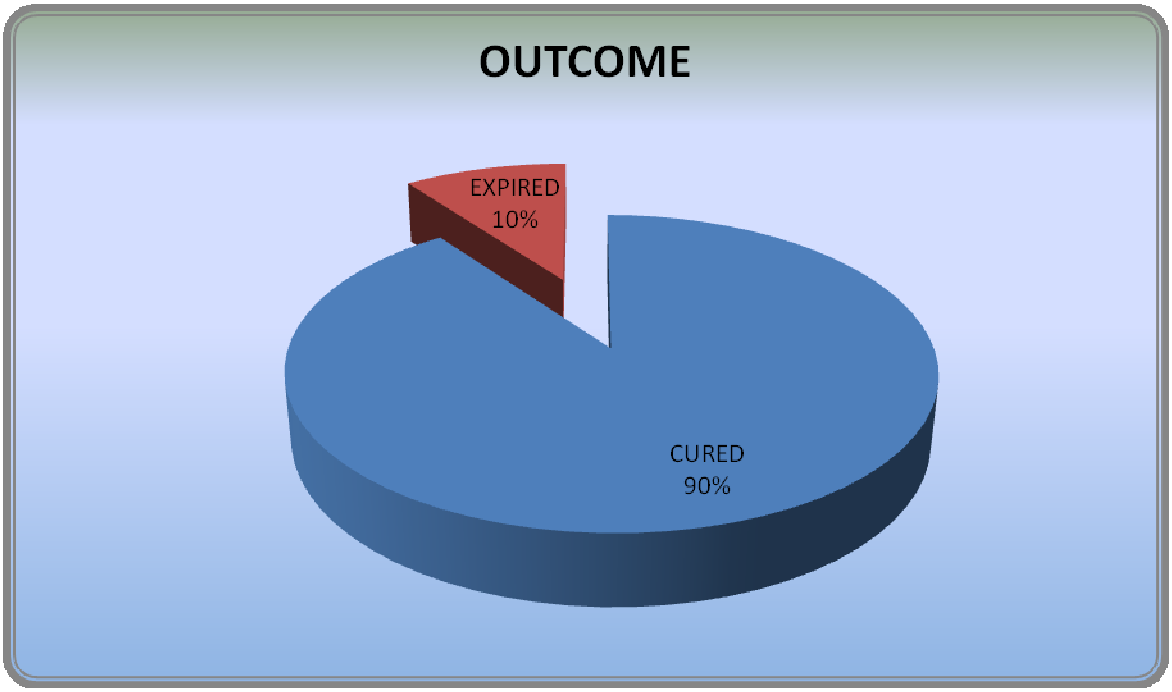


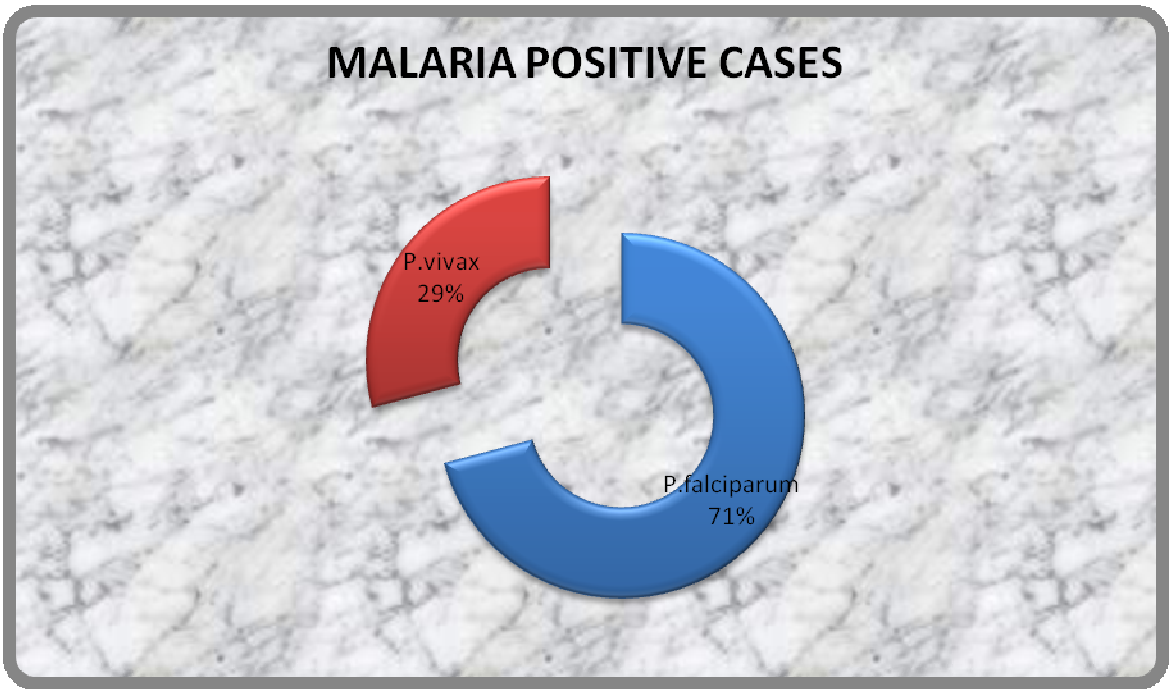

SEASONAL CLUSTERING OF CASES
NO. OF CASES
16
14
12
10
8
6
4
2
0
JANUARY
FEBRUARY
MARCH
APRIL
MAY
JUNE
JULY
AUGUST
SEPTEMBER
OCTOBER

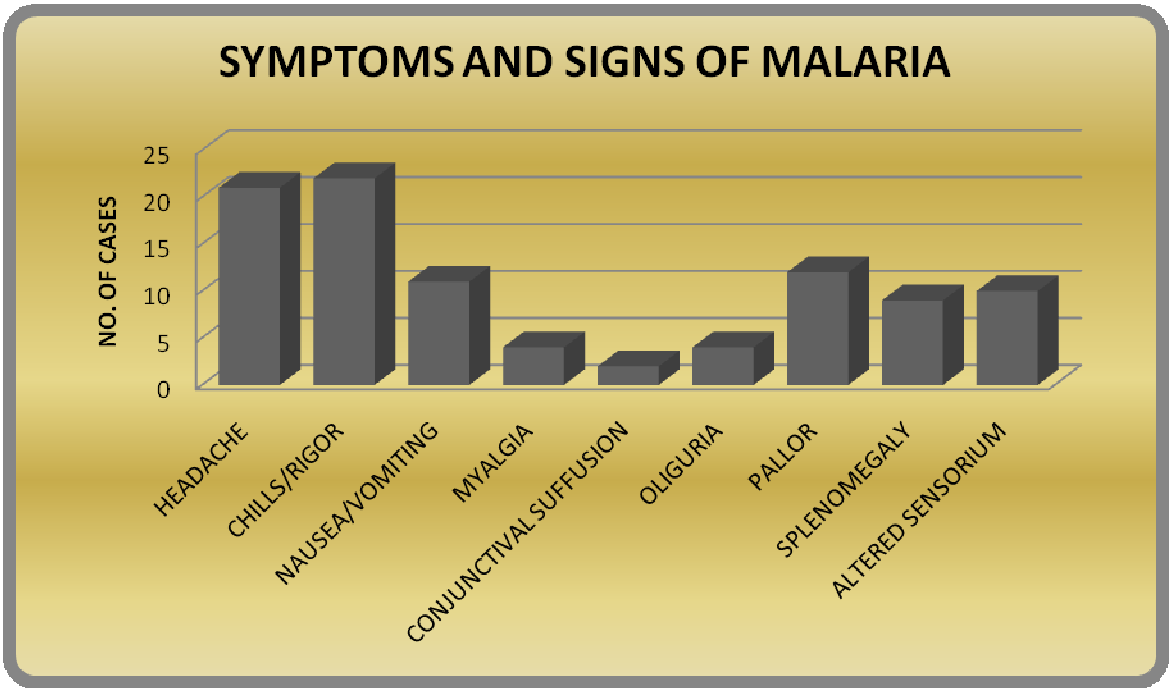

DISTRIBUTION OF SIGNS AND SYMPTOMS
50
45
40
35
30
25
20
15
10
5
0
FEVER
JAUNDICE
HEADACHE
MYALGIA
CHILLS/RIGOR
NAUSEA/VOMITING
COUGH
CONJUNCTIVAL...
ABDOMINAL PAIN
DIARRHEA
OLIGURIA
SKIN RASH
DEHYDRATION
PALLOR
PEDAL EDEMA
LYMPAHDENOPATHY
HEPATOMEGALY
SPLENOMEGALY
MUSCLE TENDERNESS
ALTERED SENSORIUM

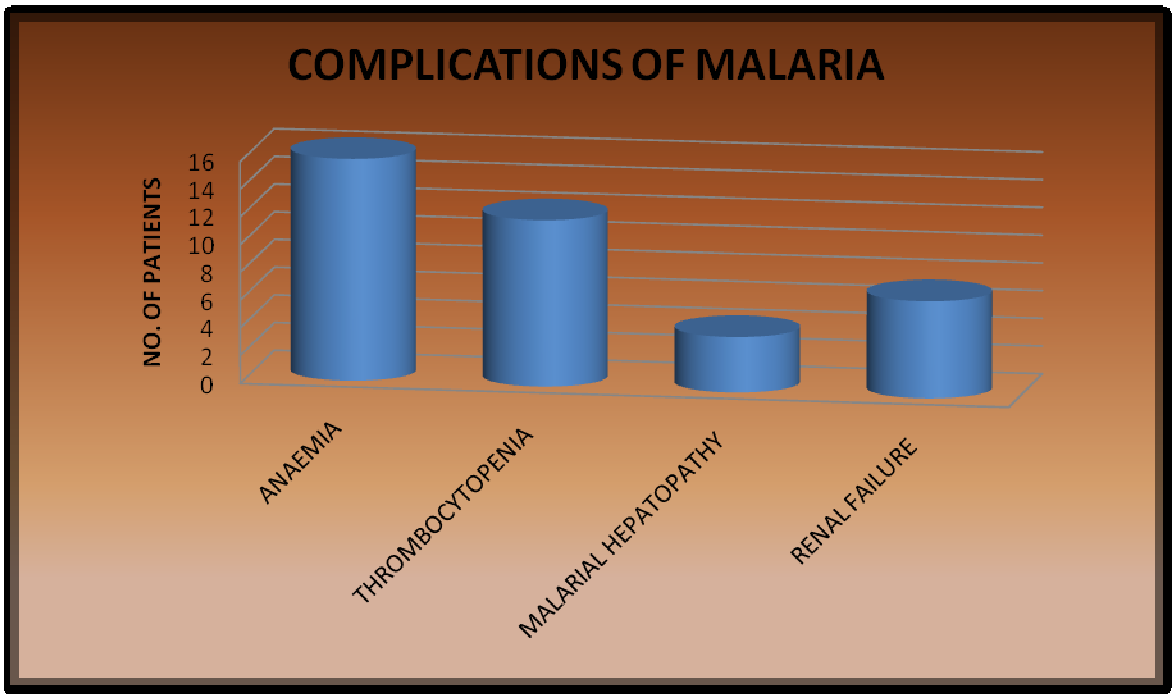

SEROVARS ANALYSIS
20
18
16
14
12
10
8
6
4
2
0
ICTERO
SEMARANGA
GRIPPOTYPHOSA
AUTOMNALIS
AUSTRALIS
NO. OF PATIENTS
20
16
6
5
3

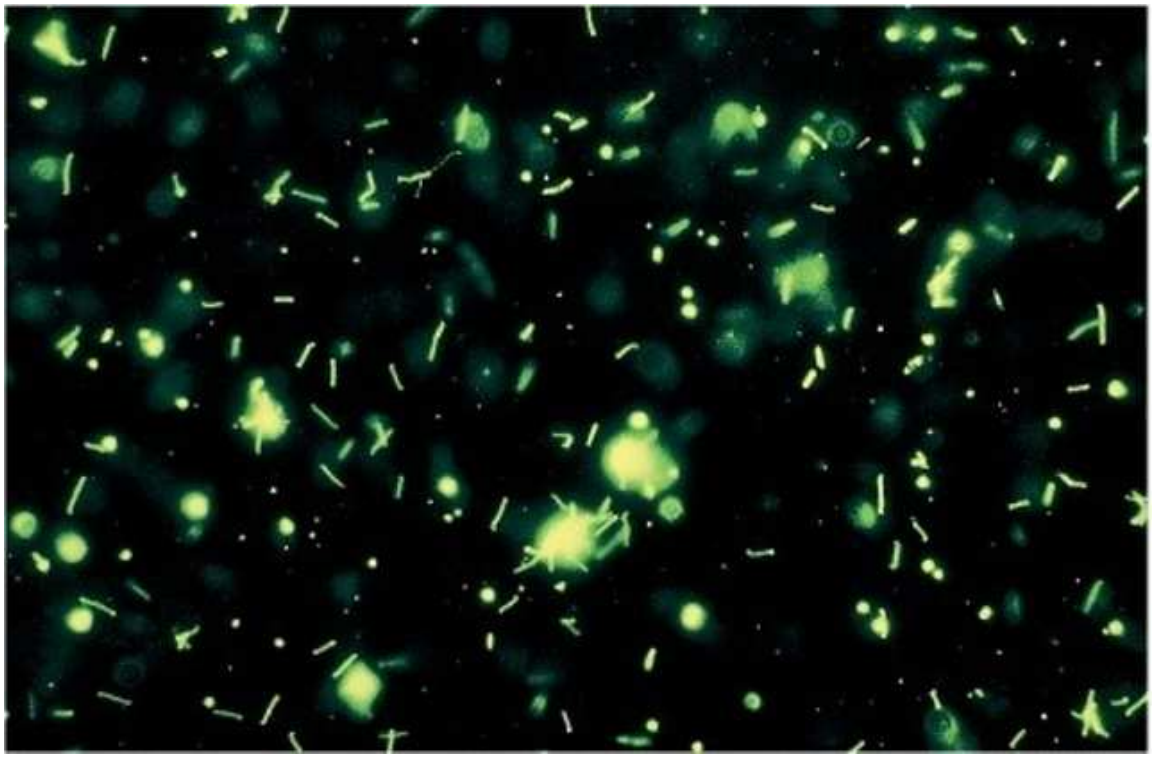

SEROVARS AND COMPLICATIONS CORRELATION
NO OF CASES
14
12
10
8
6
4
2
0
ICTERO
SEMARANGA
GRIPPOTYPHOSA
AUTOMNALIS
AUSTRALIS
THROMBOCYTOPENIA
10
4
3
3
1
RENAL FAILURE
14
0
2
2
1


OUTCOME
EXPIRED
10%
CURED
90%


MALARIA POSITIVE CASES
P.vivax
29%
P.falciparum
71%


SYMPTOMS AND SIGNS OF MALARIA
NO. OF CASES
25
20
15
10
5
0
HEADACHE
CHILLS/RIGOR
NAUSEA/VOMITING
MYALGIA
CONJUNCTIVAL SUFFUSION
OLIGURIA
PALLOR
SPLENOMEGALY
ALTERED SENSORIUM


COMPLICATIONS OF MALARIA
NO. OF PATIENTS
16
14
12
10
8
6
4
2
0
ANAEMIA
THROMBOCYTOPENIA
MALARIAL HEPATOPATHY
RENAL FAILURE



Sl. No	Name	Hb (gl)	TC (Cell)	Platted	ESR (1Hr)	TB	DB	Indirect	SGOT (U/L)
75	Nithya	12.90	4800	1.90	22.00	25.00	1.50	1.00	5240
76	Siva	11.40	8500	3.17	18.00	10.00	7.00	3.00	229
77	Raja Prasad	10.00	5800	2.10	20.00	8.00	6.20	1.80	512
78	Ravi	10.30	5800	1.60	32.00	1.80	1.20	0.60	206
79	Kamalakannan	8.90	13400	1.41	34.00	19.20	11.80	7.40	297
80	Rathan	10.00	7200	2.40	18.00	10.50	7.20	3.30	620
81	Perumal	11.70	7100	1.91	16.00	11.50	7.50	4.00	711
82	Venkatesan	13.10	6300	1.79	22.00	7.10	3.00	4.10	210
83	Ramesh	9.30	7100	1.82	32.00	6.80	4.10	2.70	221
84	Sarulatha	11.00	4800	1.72	20.00	5.80	3.60	2.20	1042
85	Karin	13.00	6400	2.40	18.00	4.00	2.00	2.00	248
86	Sundar	9.90	7200	3.10	28.00	7.20	4.10	3.10	432
87	Sathya Moorthy	12.30	11000	2.70	30.00	9.30	5.60	3.70	291
88	Sulan	11.00	9100	1.92	22.00	8.90	4.90	4.00	329
89	Nalini	10.70	8800	2.70	32.00	6.80	4.20	2.60	323
90	Saravanan	9.90	8100	3.00	20.00	6.70	4.30	2.40	212
91	Jose	10.10	7200	3.20	24.00	7.20	4.00	3.20	411
92	Palani	11.20	7300	1.79	22.00	5.30	4.00	1.30	420
93	Sudha	10.50	7200	3.40	24.00	6.30	4.10	2.20	926
94	Partion	12.10	13000	2.10	42.00	7.10	3.20	3.90	148
95	Mohan	12.70	24000	1.15	18.00	2.40	1.20	1.20	41
96	Pandi	10.00	14600	2.62	32.00	8.70	6.30	2.40	78

SGPT (U/L)	ALP	BL Urea	Sr. Creatinine (Mg/ldr)	Smear for MP	IM HAV	Hb Sag	Anti -HCV
3350.00	256.00	28.00	0.90	-	+	-	-
81.00	223.00	19.00	0.70	-	+	-	-
422.00	204.00	25.00	0.80	-	-	+	-
40.00	92.00	18.00	0.70	-	-	-	+
272.00	148.00	22.00	0.80	-	-	-	+
500.00	212.00	34.00	0.90	-	-	+	-
522.00	342.00	30.00	0.80	-	-	-	+
204.00	282.00	30.00	0.70	-	-	+	-
210.00	182.00	26.00	0.90	-	-	+	-
986.00	256.00	32.00	0.80	-	+	-	-
211.00	192.00	25.00	0.60	-	+	-	-
360.00	381.00	32.00	0.70	-	-	+	-
240.00	202.00	29.00	1.00	-	-	-	+
311.00	281.00	24.00	0.60	-	-	+	-
280.00	212.00	26.00	0.70	-	-	+	-
172.00	224.00	32.00	0.90	-	-	+	-
411.00	301.00	40.00	1.00	-	-	-	+
420.00	382.00	30.00	0.70	-	-	+	-
926.00	358.00	42.00	1.10	-	-	+	-
148.00	378.00	26.00	0.70	-	-	-	-
41.00	212.00	37.00	1.10	-	-	-	-
78.00	532.00	28.00	0.90	-	-	-	-

MSAT	MAT	Ultrasound abdomen	Diagnosis	Outcome
-	-	N	Hepatitis A	Cured
-	-	Hepatomegaly	Hepatitis A	Cured
-	-	Hepatomegaly	Hepatitis B	Cured
-	-	Hepatomegaly	Hepatitis C	Cured
-	-	Hepatomegaly	Hepatitis C	Cured
-	-	N	Hepatitis B	Cured
-	-	N	Hepatitis C	Cured
-	-	Hepatomegaly	Hepatitis B	Cured
-	-	Hepatomegaly	Hepatitis B	Cured
-	-	N	Hepatitis A	Cured
-	-	N	Hepatitis A	Cured
-	-	N	Hepatitis B	Cured
-	-	Hepatomegaly	Hepatitis C	Cured
-	-	N	Hepatitis B	Cured
-	-	Hepatomegaly	Hepatitis B	Cured
-	-	N	Hepatitis B	Cured
-	-	N	Hepatitis C	Cured
-	-	N	Hepatitis B	Cured
-	-	Hepatomegaly	Hepatitis B	Cured
-	-	Perichulic Fluid	GB Disease	Expired
-	-	CBD stone	GB Disease	Cured
-	-	CBD stone	GB Disease	Cured

MASTER CHART

Sl. No	Name	Age	Sex	Place	IP. No	Occupation	Immunisation against Hepatitis B	Duration of Fever	Duration of Jaundice	Headache	Myalgia	Chills & Rigor	Nausea/Vomiting	Cough	Conjunctival Suffusion	Abdominal pain	Diarrhea	Oliguria	Skin Rash	Dehydration	Pallor	Pedal edema	Lymadenopathy	Hepatomegaly	Splenomegaly	Muscle Tenderness	Altered Sensorium	Meningeal Sign	SCH
1	Krishnaveni	40	M	Mugappair	9949	MW		7	3	✓	✓			✓	✓		✓				✓				✓				
2	Saradha	50	F	Kanchipuram	23042	MW		12	2	✓	✓		✓		✓		✓	✓		✓		✓				✓	✓		
3	Vengat	38	M	Andra	7292	NMW		15	5	✓	✓	✓		✓		✓										✓	✓		
4	Vivek	13	M	Kodambakkam	10914	S		8	4	✓	✓		✓		✓					✓	✓						✓		
5	Durai Singh	60	M	Mambalam	23192	MW		9	5	✓	✓		✓	✓	✓		✓			✓						✓			✓
6	Ezhumalai	70	M	Vadapalani	47129	MW		11	7		✓	✓		✓		✓					✓								
7	Varadharaj	25	M	Arakonam	60181	NMW		13	10	✓	✓		✓		✓											✓	✓		
8	Siranjeevi	21	M	Thiruvallur	63838	NMW		12	7		✓			✓		✓	✓	✓	✓						✓		✓		
9	Sarangan	70	M	Kanchipuram	74887	NMW		7	2		✓		✓		✓						✓			✓		✓			
10	Suresh Kumar	44	M	Perambur	62198	MW		9	4	✓	✓		✓		✓	✓				✓									
11	Jaya	25	F	Manali	47282	O		8	3	✓	✓	✓		✓		✓				✓						✓	✓		
12	Batcha	34	M	Saidapet	23492	MW		6	2		✓			✓	✓		✓				✓								
13	Nadhan	48	M	Kodambakkam	42942	MW		7	1				✓		✓											✓	✓		
14	Stella Mary	29	F	Viyasarpadi	42492	O		6	3	✓	✓		✓		✓		✓				✓	✓					✓		
15	Neetu Yadav	55	M	Perambur	64948	MW		9	4	✓	✓	✓			✓			✓		✓						✓			
16	Johnson	48	M	Koungaiyur	49212	MW		10	5		✓		✓			✓		✓						✓		✓	✓		
17	Naresh	31	M	Washermenpet	41911	MW		12	7	✓	✓		✓		✓						✓					✓	✓		
18	Rohitha	49	F	Ennore	31590	NMW		12	6		✓	✓		✓	✓												✓		
19	Aswin	40	F	Egmore	32913	MW		11	4		✓		✓																
20	Nallasivam	41	M	Thiruvetriyur	33579	MW		7	3	✓	✓				✓							✓					✓		
21	Jayalakshmi	53	F	Washermenpet	33515	MW		5	2		✓		✓		✓					✓						✓			
22	Vijayanthi	25	F	Washermenpet	33695	NMW		8	4		✓	✓		✓										✓					
23	Harish	15	M	Kilpauk	33714	S		11	5		✓				✓		✓				✓								✓
24	Ismail	40	M	Tiruvottiyur	33777	MW		10	7		✓		✓		✓											✓			

Sl. No	Name	Age	Sex	Place	IP. No	Occupation	Immunisation against Hepatitis B	Duration of Fever	Duration of Jaundice	Headache	Myalgia	Chills & Rigor	Nausea/Vomiting	Cough	Conjunctival Suffusion	Abdominal pain	Diarrhea	Oliguria	Skin Rash	Dehydration	Pallor	Pedal edema	Lymadenopathy	Hepatomegaly	Splenomegaly	Muscle Tenderness	Altered Sensorium	Meningeal Sign	SCH
25	Immanuel	17	M	Rayapuram	73668	O		12	8		✓			✓	✓														
26	Kumara Raja	39	M	Kilpauk	73659	MW		6	2	✓	✓		✓		✓		✓				✓								
27	Gnanasundari	34	F	Washermenpet	64231	MW		9	4	✓	✓	✓		✓		✓						✓				✓			
28	Ayyadurai	44	M	Vadapalani	72129	MW		11	5	✓	✓		✓		✓					✓							✓		
29	Asaithambi	17	M	Redhills	81211	S		10	7			✓		✓	✓						✓			✓	✓	✓			
30	Sudha	38	F	Kodambakkam	51418	MW		12	7	✓	✓		✓		✓					✓		✓							
31	Saritha	27	F	Choolai	72698	O		7	2	✓	✓				✓		✓									✓	✓		
32	Leela Rani	47	F	Ennore	28555	MW		14	8			✓		✓	✓								✓						
33	Sampath	27	M	Royapuram	30938	MW		7	3	✓	✓			✓		✓						✓							
34	Loganathan	40	M	Thiruvotriyur	90111	MW		9	4	✓	✓	✓		✓	✓									✓					
35	Arumugam	35	M	Egmore	43129	MW		8	4	✓	✓		✓		✓		✓								✓	✓			
36	Kumar	39	M	Saidapet	72121	MW		10	5	✓	✓			✓		✓				✓									
37	Thusif	43	M	Ennore	94312	NMW		12	7	✓	✓	✓	✓		✓									✓					
38	Rajan	55	M	Tondayarpet	74521	MW		7	3		✓				✓		✓	✓				✓				✓			
39	Naveen Kumar	14	M	Washermenpet	85241	S		10	4	✓	✓			✓		✓			✓										
40	Priskilla	17	F	Porur	74213	O		15	11	✓	✓	✓			✓												✓		✓
41	Manoj	36	M	Royapuram	85942	MW		13	10	✓	✓		✓		✓					✓		✓				✓			
42	Sidharthan	40	M	Manali	74211	MW		14	12		✓			✓	✓														
43	Rani	15	F	Thiruvotriyur	94223	O		8	4		✓				✓			✓			✓			✓					
44	Veeraragavan	54	M	Chennai GPO	74312	MW		7	2	✓	✓				✓											✓			
45	Thanjammal	21	F	Porur	92411	NMW		11	7	✓	✓				✓							✓							
46	Logammal	17	F	Tondayarpet	92142	O		5	1	✓	✓				✓		✓				✓			✓					
47	Shyam Kumar	13	M	Guindy	73149	O		7	4	✓	✓	✓			✓														
48	Jegan	24	M	Anna Nagar	73227	MW		9	5	✓	✓				✓							✓							
49	Narayanan	47	M	Mogapair	82723	MW		10	7		✓		✓					✓		✓				✓		✓			
50	Parvathy	36	F	Washermenpet	94518	MW		11	3	✓	✓				✓						✓			✓					

Sl. No	Name	Age	Sex	Place	IP. No	Occupation	Immunisation against Hepatitis B	Duration of Fever	Duration of Jaundice	Headache	Myalgia	Chills & Rigor	Nausea/Vomiting	Cough	Conjunctival Suffusion	Abdominal pain	Diarrhea	Oliguria	Skin Rash	Dehydration	Pallor	Pedal edema	Lymadenopathy	Hepatomegaly	Splenomegaly	Muscle Tenderness	Altered Sensorium	Meningeal Sign	SCH
51	Parimala	39	F	Egmore	6352	O		4	2	✓		✓	✓								✓								
52	Nagaraju	45	M	Anna Nagar	22944	MW		6	3	✓	✓	✓													✓				
53	Jayalakshmi	31	F	Parrys	59077	NMW		5	3	✓		✓	✓								✓						✓		
54	Sivakumar	32	M	Velachery	74906	NMW		3	2	✓		✓	✓								✓				✓				
55	Udayakumar	40	M	Vadapalani	49239	MW		7	4	✓		✓						✓		✓	✓	✓					✓		
56	Vijay	17	M	Redhills	74239	MW		4	3	✓	✓	✓	✓											✓			✓		
57	Prema	42	F	Ennore	82343	MW		5	2	✓		✓	✓		✓					✓	✓						✓		
58	Muthu	37	M	Arakonam	72491	NMW		5	4			✓													✓				
59	Vinnai	49	M	Saidapet	74289	MW		7	5	✓		✓				✓					✓								
60	Kumar	32	M	SIET	73921	NMW		3	2	✓	✓		✓							✓									
61	Kala	29	F	Chrompet	82911	O		6	4	✓		✓	✓														✓		
62	Subramani	40	M	Guindy	92129	NMW		7	4	✓		✓	✓			✓										✓			
63	Anand	39	M	Brodway	72193	MW		6	5	✓	✓	✓		✓				✓		✓	✓	✓							
64	Babu	21	M	Thiruvetriyur	82144	S		5	3	✓		✓																	
65	Santhose	43	M	Kanchipuram	92311	MW		6	4			✓	✓		✓														
66	Priya	34	F	Anna Nagar	84429	NMW		8	5	✓		✓	✓			✓		✓			✓				✓		✓		
67	Leenus	49	M	Chengalpet	92484	MW		4	2	✓																			
68	Prema	28	F	Redhills	24948	NMW		3	3	✓		✓									✓			✓					
69	Murali	17	M	Tambaram	42913	S		7	4	✓		✓															✓		
70	Kannan	29	M	Guindy	92144	MW		3	3			✓	✓								✓								
71	Jeya	51	F	Kilpauk	42001	MW		7	4	✓		✓									✓								
72	Kanmani	41	F	Anna Nagar	42103	NMW		9	6	✓		✓						✓			✓						✓		
73	Arumugam	56	M	Amjekarai	92193	O		4	2	✓		✓															✓		
74	Sudhakar	35	M	Porur	93281	MW		5	3	✓		✓												✓			✓		
75	Nithya	23	F	Park Town	92319	S	✓	7	3	✓	✓		✓			✓													
76	Sivakumar	18	M	Chrompet	98844	S		4	4				✓											✓					

Sl. No	Name	Age	Sex	Place	IP. No	Occupation	Immunisation against Hepatitis B	Duration of Fever	Duration of Jaundice	Headache	Myalgia	Chills & Rigor	Nausea/Vomiting	Cough	Conjunctival Suffusion	Abdominal pain	Diarrhea	Oliguria	Skin Rash	Dehydration	Pallor	Pedal edema	Lymadenopathy	Hepatomegaly	Splenomegaly	Muscle Tenderness	Altered Sensorium	Meningeal Sign	SCH
77	Raja Prasad	17	M	Tambaram	8219	MW		15	15			✓	✓																
78	Ravi	44	M	Vennarpet	60278	NMW		5	5	✓	✓																		
79	Kamalakannan	68	M	Kodambakkam	63976	MW		3	3															✓					
80	Rathan	39	M	Porur	73142	MW		8	7				✓																
81	Perumal	49	M	Arakonam	74192	MW		4	3				✓																
82	Venkatesan	38	M	Porur	74093	MW		9	5				✓											✓					
83	Ramesh	40	M	Kadpadi	72432	MW		5	5		✓	✓	✓							✓									
84	Sarulatha	51	F	Redhills	78341	MW		7	3							✓		✓											
85	Kavin	17	M	MMDA	87431	S		8	4		✓	✓	✓						✓										
86	Sundar	28	M	SIET	78329	NMW		4	4	✓		✓	✓																
87	Sathymoorthy	47	M	Chrompet	48494	MW		7	6		✓	✓	✓			✓				✓									
88	Sulan	24	M	Kilpauk	74921	NMW		6	6																				
89	Nalini	32	F	Egmore	78291	MW		5	3		✓	✓	✓			✓		✓											
90	Saravanan	40	M	Saligramam	74282	NMW		4	4																				
91	Jose	47	M	Redhills	74884	MW		7	7	✓	✓	✓	✓			✓													
92	Palani	37	M	Egmore	82197	NMW		8	4		✓	✓	✓							✓									
93	Sudha	29	F	Vadapalani	81429	MW		9	5	✓						✓													
94	Parthiban	27	M	Tiruvallur	74889	NMW		5	5	✓	✓	✓	✓			✓	✓			✓									
95	Mohan	37	M	Gummidipondi	58224	MW		7	7								✓			✓									
96	Pandi	30	M	Arakonam	58763	MW		15	15			✓	✓				✓												

N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Fundus

N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Fundus

																								Fundus

																		Fundus

Sl. No	Name	Hemogram				Liver Function Test						BL Urea	Sr. Creatinine (Mg/dl)	Smear for MP	IgM-HAV	Hb sAg	Anti -HCV	MSAT	MAT	Ultrasound abdomen	Diagnosis	Outcome
		Hb (gm/dl)	TC (Cells/ml/µl)	Platelet Count	ESR (1Hr)	TB (mg/dl)	Direct Bilirubin	Indirect Bilirubin	SGOT (U/L)	SGPT (U/L)	ALP											
1	Krishnaveni	7.1	4100	0.87	120	9.3	4.3	5.2	35	26	82	24	0.7	Neg	Neg	Neg	Neg	2+	Ictero	Splenomegaly	Lepto	Cured
2	Saradha	10.2	8900	1	40	38	28	10	168	76	122	174	2.9	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Expired
3	Vengat	10.2	6000	1	24	46	1.6	3	80	82	120	25	0.8	Neg	Neg	Neg	Neg	2+	Autumnalis	Hepatomegaly	Lepto	Cured
4	Vivek	8.4	7000	1	60	4.9	3.2	1.7	71	62	183	30	0.9	Neg	Neg	Neg	Neg	2+	Grippotyphosa	N	Lepto	Cured
5	Durai Singh	13.4	4400	0.31	10	2.1	1.5	0.6	58	82	169	38	0.8	Neg	Neg	Neg	Neg	3+	Ictero	N	Lepto	Cured
6	Ezhumalai	8.4	6400	0.9	58	5.1	2.3	2.8	38	46	172	90	1.2	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
7	Varadharaj	11.1	6700	1.11	30	3	1.6	1.4	132	117	130	27	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
8	Siranjeevi	13.1	7100	3.33	6	6	3.4	2.6	75	55	88	28	1.0	Neg	Neg	Neg	Neg	2+	Autumnalis	Splenomegaly	Lepto	Cured
9	Sarangan	9.2	11000	0.5	32	2.2	1.8	0.4	58	42	132	180	8.0	Neg	Neg	Neg	Neg	3+	Ictero	N	Lepto	Expired
10	Suresh Kumar	11.3	12000	1.17	12	4.6	3	1.6	80	74	123	72	2.7	Neg	Neg	Neg	Neg	3+	Ictero	Hepatomegaly	Lepto	Cured
11	Jaya	12.1	7200	2.12	6	25.2	18	7.2	73	86	190	38	0.8	Neg	Neg	Neg	Neg	2+	Australis	N	Lepto	Cured
12	Batcha	9.4	8100	1.1	24	7.2	4.1	3.1	92	90	212	28	0.7	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
13	Nadhan	10.5	4230	1	36	4	2	2	48	45	120	42	1.1	Neg	Neg	Neg	Neg	3+	Ictero	N	Lepto	Cured
14	Stella Mary	8.9	5100	1.54	30	8.4	4	4.4	76	80	138	28	0.8	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
15	Neetu Yadav	11.4	4720	1.32	30	12.5	8	4.5	120	132	198	91	3.9	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
16	Johnson	13.4	5200	1.78	42	20.1	10	10.1	212	300	212	140	7.0	Neg	Neg	Neg	Neg	2+	Ictero	Hepatomegaly	Lepto	Expired
17	Naresh	8.4	5400	1.62	28	12.4	6.8	5.6	110	112	162	112	4.2	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
18	Rohitha	9.9	7100	2.42	14	3	1.6	1.4	70	70	128	36	9.0	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
19	Aswin	10.5	6400	0.48	8	10.5	6.3	4.2	108	110	142	32	1.1	Neg	Neg	Neg	Neg	2+	Autumnalis	N	Lepto	Cured
20	Nallasivam	11	7200	2.32	12	8.1	3.9	4.2	112	120	300	40	1.2	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
21	Jayalakshmi	10.7	5200	1.48	32	4.7	2.7	2	42	48	92	35	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
22	Vijayanthi	11.3	4900	1.62	26	6	3.2	2.8	52	48	112	37	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	Hepatomegaly	Lepto	Cured
23	Harish	9.6	5400	1.32	18	7.2	4.1	3.1	68	72	120	28	0.6	Neg	Neg	Neg	Neg	2+	Grippotyphosa	N	Lepto	Cured
24	Ismail	10.3	7200	3	42	6.4	3.6	2.8	72	78	116	34	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured

Sl. No	Name	Hemogram				Liver Function Test						BL Urea	Sr. Creatinine (Mg/dl)	Smear for MP	IgM-HAV	Hb sAg	Anti -HCV	MSAT	MAT	Ultrasound abdomen	Diagnosis	Outcome
		Hb (gm/dl)	TC (Cells/ml/µl)	Platelet Count	ESR (1Hr)	TB (mg/dl)	Direct Bilirubin	Indirect Bilirubin	SGOT (U/L)	SGPT (U/L)	ALP											
25	Immanuel	10.4	7200	1.92	12	12.2	6.2	6	88	120	148	36	0.9	Neg	Neg	Neg	Neg	2+	Australis	N	Lepto	Cured
26	Kumara Raja	8.2	4100	1	12	8.6	4	4.6	50	45	96	92	3.6	Neg	Neg	Neg	Neg	3+	Ictero	N	Lepto	Cured
27	Gnanasundari	11.2	7200	1.72	32	14.2	8.4	5.8	40	36	112	40	1.0	Neg	Neg	Neg	Neg	2+	Grippotyphosa	N	Lepto	Cured
28	Ayyadurai	11.9	6100	1.68	28	4.2	2	2.2	52	48	102	36	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
29	Asaithambi	9.0	7200	1.9	16	6.8	4.2	2.6	46	50	96	29	0.7	Neg	Neg	Neg	Neg	2+	Ictero	Hepatosplenomegaly	Lepto	Cured
30	Sudha	10.0	6800	1.21	40	11.2	6.2	5	240	232	180	112	3.0	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
31	Saritha	11.2	11000	2.4	18	11.4	6.1	5.3	40	36	126	68	2.1	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
32	Leela Rani	11.4	10500	0.9	30	10.2	5.4	4.8	72	64	112	74	2.4	Neg	Neg	Neg	Neg	3+	Autumnalis	N	Lepto	Cured
33	Sampath	12.0	10000	2.8	24	4.8	3	1.8	90	92	140	39	0.9	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
34	Loganathan	10.8	6000	3.3	18	9.2	4.1	5.1	40	41	84	30	0.7	Neg	Neg	Neg	Neg	2+	Semaranga	Hepatomegaly	Lepto	Cured
35	Arumugam	14.0	7100	1.4	32	4.2	2.1	2.1	40	38	176	48	2.0	Neg	Neg	Neg	Neg	2+	Australis	Splenomegaly	Lepto	Cured
36	Kumar	12.0	7600	1.9	18	6.8	5	1.8	42	36	152	40	1.1	Neg	Neg	Neg	Neg	2+	Grippotyphosa	N	Lepto	Cured
37	Thusif	11.4	4800	2.4	36	11.2	7.1	4.1	40	44	86	80	3.2	Neg	Neg	Neg	Neg	3+	Ictero	Hepatomegaly	Lepto	Cured
38	Rajan	11.7	5100	2.2	8	10.4	5.8	4.6	42	40	92	128	6.1	Neg	Neg	Neg	Neg	3+	Ictero	N	Lepto	Expired
39	Naveen Kumar	10.2	5400	1.9	42	6.8	3.8	3	40	36	106	37	1.0	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
40	Priskilla	10.8	4900	0.31	40	5.4	3.4	2	42	38	112	54	1.7	Neg	Neg	Neg	Neg	2+	Grippotyphosa	N	Lepto	Cured
41	Manoj	10.0	5600	2.3	50	3.8	1.8	2	54	50	86	36	0.8	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
42	Sidharthan	13.0	8900	1.7	16	8.4	5.1	3.3	198	196	240	28	0.6	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
43	Rani	7.4	5900	1.7	8	10.2	5.1	5.1	40	36	118	70	2.8	Neg	Neg	Neg	Neg	3+	Ictero	Hepatomegaly	Lepto	Cured
44	Veeraragavan	10.0	7200	1.5	6	3.2	1.1	2.1	42	40	96	28	0.7	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
45	Thanjammal	10.8	6000	1.9	12	4.2	2.8	1.4	38	45	64	71	3.1	Neg	Neg	Neg	Neg	2+	Autumnalis	N	Lepto	Expired
46	Logammal	8.4	7100	2	16	6.4	3	3.4	50	56	102	65	1.5	Neg	Neg	Neg	Neg	2+	Grippotyphosa	Hepatomegaly	Lepto	Cured
47	Shyam Kumar	10.2	4300	2.4	18	4.2	2.1	2.1	62	64	92	40	1.0	Neg	Neg	Neg	Neg	2+	Ictero	N	Lepto	Cured
48	Jegan	11.0	4000	2.6	32	3.8	2.1	1.7	40	36	112	29	1.0	Neg	Neg	Neg	Neg	2+	Semaranga	Hepatomegaly	Lepto	Cured

Sl. No	Name	Hemogram				Liver Function Test						BL Urea	Sr. Creatinine (Mg/dl)	Smear for MP	IgM-HAV	Hb sAg	Anti -HCV	MSAT	MAT	Ultrasound abdomen	Diagnosis	Outcome
		Hb (gm/dl)	TC (Cells/ml/µl)	Platelet Count	ESR (1Hr)	TB (mg/dl)	Direct Bilirubin	Indirect Bilirubin	SGOT (U/L)	SGPT (U/L)	ALP											
49	Narayanan	11.0	5300	3	28	6.6	3.6	3	70	72	122	72	1.9	Neg	Neg	Neg	Neg	2+	Ictero	Hepatomegaly	Lepto	Cured
50	Parvathy	7.6	5500	1	16	4.2	2.2	2	84	90	142	35	0.7	Neg	Neg	Neg	Neg	2+	Semaranga	N	Lepto	Cured
51	Parimala	6.5	29800	1	28	6.6	5.2	1.4	80	56	102	144	3.8	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
52	Nagaraju	10.5	3100	0.9	32	2	9.5	10.5	290	226	346	114	3.5	PF	Neg	Neg	Neg	Neg	Neg	Hepatosplenomegaly	Malaria	Expired
53	Jayalakshmi	9.1	8100	2.1	64	1.8	1.5	0.3	82	95	128	26	0.6	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
54	Sivakumar	10.6	6000	0.65	24	5.2	1.5	3.7	30	73	110	70	1.4	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
55	Udayakumar	7.6	9100	1.2	22	4	2	2	42	45	122	36	0.8	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
56	Vijay	11	7900	2.1	28	2.2	1	1.2	40	47	110	37	0.7	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
57	Prema	7.6	5300	0.91	22	6.8	1.8	5	221	248	278	82	2.7	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
58	Muthu	11.7	9100	2.7	28	3.4	1.4	2	62	67	129	40	1.1	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
59	Vinnai	13	9000	0.95	32	2.1	1	1.1	39	43	109	32	0.7	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
60	Kumar	7.9	4700	1.27	24	3.1	1	2.1	49	40	123	48	1.2	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
61	Kala	11.2	6900	1.58	32	2.4	1.2	1.2	38	43	120	28	0.7	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
62	Subramani	7.4	5300	1.72	26	4.9	2.4	2.5	62	78	149	61	1.4	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
63	Anand	12.7	9300	1.98	40	2.9	1.4	1.5	32	38	98	28	0.6	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
64	Babu	8.4	4900	1.4	16	9	3	6	291	220	194	49	1.9	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
65	Santhose	7	4100	1.2	24	4.3	1.3	3	92	80	162	42	1.1	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
66	Priya	8.2	7300	2.2	24	3.1	1.1	2	49	42	134	36	0.9	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
67	Leenus	11	7900	3.1	32	2.4	1.3	1.1	38	32	190	28	0.6	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
68	Prema	8.6	4300	0.75	18	3.4	1.1	2.3	43	43	129	32	0.8	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
69	Murali	13.2	11000	1.93	24	2.7	1.3	1.4	28	37	120	26	0.7	PV	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
70	Kannan	9.1	5900	1.12	28	3.1	1.2	1.9	48	43	136	32	0.8	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
71	Jeya	8.9	7200	2.1	32	4	2.1	1.9	57	51	126	39	0.9	PV	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	cured
72	Kanmani	7.6	5900	1.2	24	7.3	2.3	5	262	212	249	72	2.4	PF	Neg	Neg	Neg	Neg	Neg	Splenomegaly	Malaria	Expired

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		Hb (gm/dl)	TC (Cells/ml/µl)	Platelet Count	ESR (1Hr)	TB (mg/dl)	Direct Bilirubin	Indirect Bilirubin	SGOT (U/L)	SGPT (U/L)	ALP											
73	Arumugam	11.2	9300	1.97	28	4	2	2	42	43	149	36	0.8	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
74	Sudhakar	10.1	4000	2.9	26	2.6	0.8	1.8	51	43	103	28	0.7	PF	Neg	Neg	Neg	Neg	Neg	N	Malaria	cured
75	Nithya	12.90	4800	1.90	22.00	25.00	1.50	1.00	5240	3350	256	28	0.90	Neg	+	Neg	Neg	Neg	Neg	N	Hepatitis A	Cured
76	Siva	11.40	8500	3.17	18.00	10.00	7.00	3.00	229	81	223	19	0.70	Neg	+	Neg	Neg	Neg	Neg	Hepatomegaly	Hepatitis A	Cured
77	Raja Prasad	10.00	5800	2.10	20.00	8.00	6.20	1.80	512	422	204	25	0.80	Neg	Neg	+	Neg	Neg	Neg	Hepatomegaly	Hepatitis B	Cured
78	Ravi	10.30	5800	1.60	32.00	1.80	1.20	0.60	206	40	92	18	0.70	Neg	Neg	Neg	+	Neg	Neg	Hepatomegaly	Hepatitis C	Cured
79	Kamalakannan	8.90	13400	1.41	34.00	19.20	11.80	7.40	297	272	148	22	0.80	Neg	Neg	Neg	+	Neg	Neg	Hepatomegaly	Hepatitis C	Expired
80	Rathan	10.00	7200	2.40	18.00	10.50	7.20	3.30	620	500	212	34	0.90	Neg	Neg	+	Neg	Neg	Neg	N	Hepatitis B	Cured
81	Perumal	11.70	7100	1.91	16.00	11.50	7.50	4.00	711	522	342	30	0.80	Neg	Neg	Neg	+	Neg	Neg	N	Hepatitis C	Cured
82	Venkatesan	13.10	6300	1.79	22.00	7.10	3.00	4.10	210	204	282	30	0.70	Neg	Neg	+	Neg	Neg	Neg	Hepatomegaly	Hepatitis B	Cured
83	Ramesh	9.30	7100	1.82	32.00	6.80	4.10	2.70	221	210	182	26	0.90	Neg	Neg	+	Neg	Neg	Neg	Hepatomegaly	Hepatitis B	Cured
84	Sarulatha	11.00	4800	1.72	20.00	5.80	3.60	2.20	1042	986	256	32	0.80	Neg	+	Neg	Neg	Neg	Neg	N	Hepatitis A	Cured
85	Karin	13.00	6400	2.40	18.00	4.00	2.00	2.00	248	211	192	25	0.60	Neg	+	Neg	Neg	Neg	Neg	N	Hepatitis A	Cured
86	Sundar	9.90	7200	3.10	28.00	7.20	4.10	3.10	432	360	381	32	0.70	Neg	Neg	+	Neg	Neg	Neg	N	Hepatitis B	Cured
87	Sathya Moorthy	12.30	11000	2.70	30.00	9.30	5.60	3.70	291	240	202	29	1.00	Neg	Neg	Neg	+	Neg	Neg	Hepatomegaly	Hepatitis C	Cured
88	Sulan	11.00	9100	1.92	22.00	8.90	4.90	4.00	329	311	281	24	0.60	Neg	Neg	+	Neg	Neg	Neg	N	Hepatitis B	Cured
89	Nalini	10.70	8800	2.70	32.00	6.80	4.20	2.60	323	280	212	26	0.70	Neg	Neg	+	Neg	Neg	Neg	Hepatomegaly	Hepatitis B	Cured
90	Saravanan	9.90	8100	3.00	20.00	6.70	4.30	2.40	212	172	224	32	0.90	Neg	Neg	+	Neg	Neg	Neg	N	Hepatitis B	Cured
91	Jose	10.10	7200	3.20	24.00	7.20	4.00	3.20	411	411	301	40	1.00	Neg	Neg	Neg	+	Neg	Neg	N	Hepatitis C	Cured
92	Palani	11.20	7300	1.79	22.00	5.30	4.00	1.30	420	420	382	30	0.70	Neg	Neg	+	Neg	Neg	Neg	N	Hepatitis B	Cured
93	Sudha	10.50	7200	3.40	24.00	6.30	4.10	2.20	926	926	358	42	1.10	Neg	Neg	+	Neg	Neg	Neg	Hepatomegaly	Hepatitis B	Cured
94	Partion	12.10	13000	2.10	42.00	7.10	3.20	3.90	148	148	378	26	0.70	Neg	Neg	Neg	Neg	Neg	Neg	Pericholic Fluid	GB Disease	Expired
95	Mohan	12.70	24000	1.15	18.00	2.40	1.20	1.20	41	41	212	37	1.10	Neg	Neg	Neg	Neg	Neg	Neg	CBD stone	GB Disease	Cured
96	Pandi	10.00	14600	2.62	32.00	8.70	6.30	2.40	78	78	532	28	0.90	Neg	Neg	Neg	Neg	Neg	Neg	CBD stone	GB Disease	Cured